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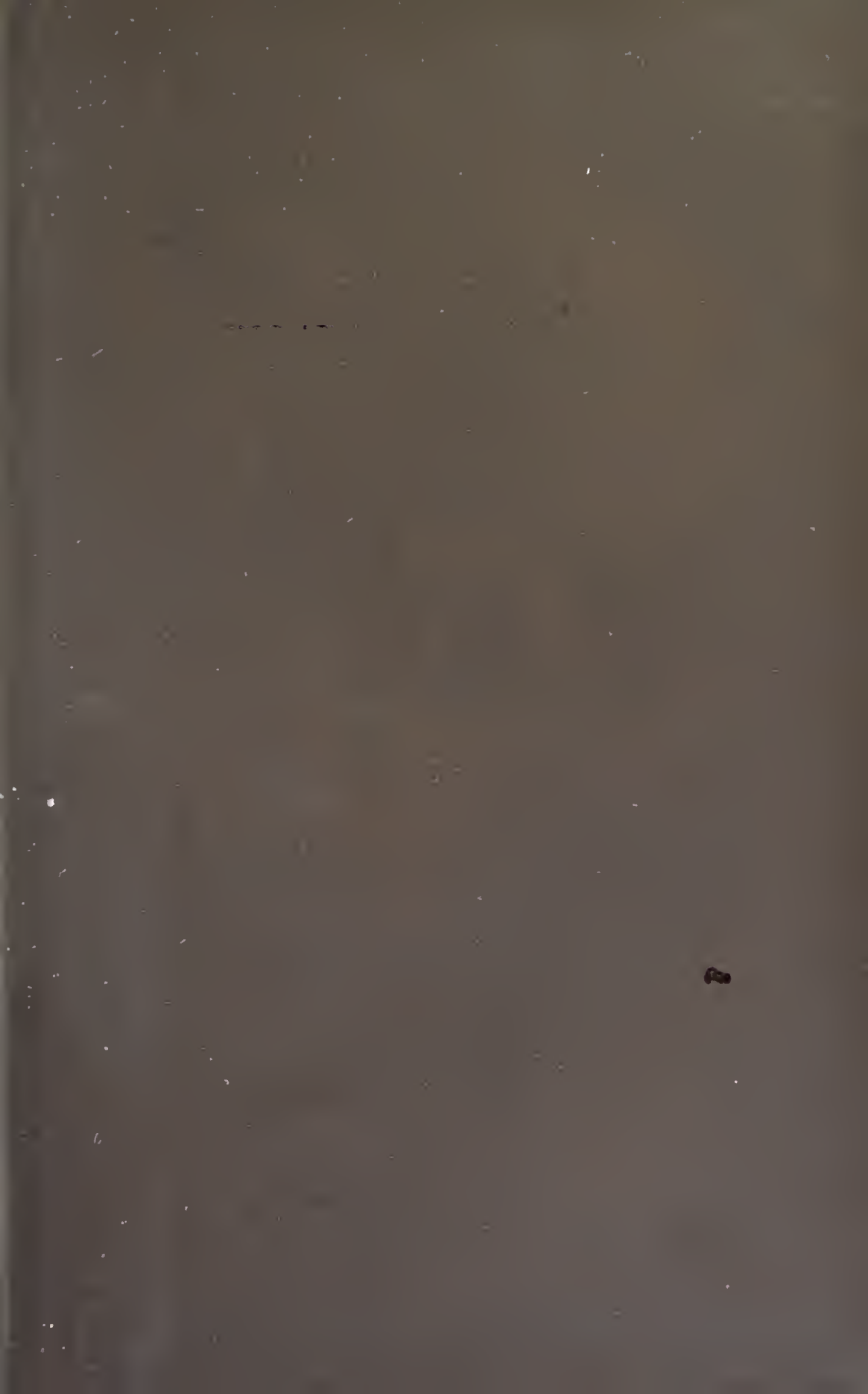
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ESSAYS IN  
HEART AND LUNG DISEASE





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HEART AND LUNG  
DISEASE

BY

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*To*  
THE STUDENTS  
OF  
THE BIRMINGHAM SCHOOL  
FROM WHOM I HAVE LEARNED  
SO MUCH  
*These Essays*  
ARE  
AFFECTIONATELY INSCRIBED





*O happy he who can the cause of Ill discern!  
Yet happier still, if he that Cause away may turn.  
Such joys doth Medicine—great Mistress—ever give  
To them who stedfastly her true Disciples live.*

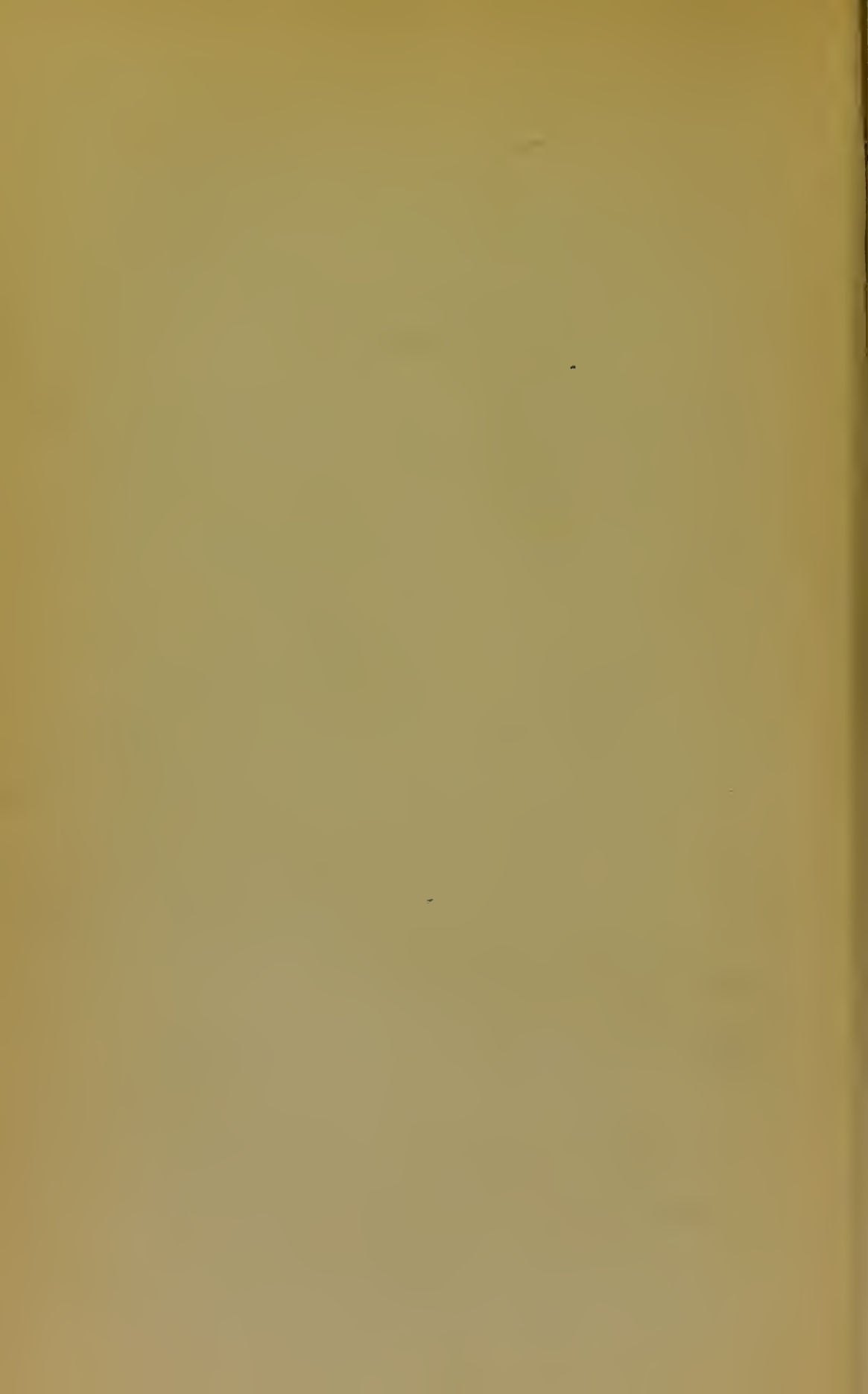


## PREFACE

*THE greater part of these Essays has been read before various medical societies during the last ten years and most of them have found their way into print ; but for the purposes of this volume all have been carefully revised and much fresh matter added. They thus accurately represent my present views on the topics treated, and, where mine differ in any way from those generally held, I have endeavoured to clearly indicate the extent of such difference. I have also tried by continuous acquaintance with current literature to omit no new knowledge which seemed to me pertinent to the subjects dealt with, which effort has necessitated much re-writing, with the result, I fear, of sometimes detracting from any literary value the original Essays may have possessed.*

ARTHUR FOXWELL.

BIRMINGHAM, May 1895.





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# ESSAYS IN HEART AND LUNG DISEASE

## I. ON DYSPNŒA

MR. PRESIDENT AND GENTLEMEN,—The title of my paper is a very wide one : of this I was well aware when I submitted it to Mr. Pritchett as a suitable subject for discussion here : but when I came to look through some of the literature relating to it, its wideness struck me as being greater even than I had anticipated : I find it mentioned as a prominent symptom in some 140 different conditions of disease.

This knowledge has only confirmed me in my previous determination to confine myself to such forms of dyspnœa as I have had good experience of, and, moreover, have felt special interest in.

You will pardon me then, if I pay more attention to the functional and perhaps minor causes of this great symptom.

The reasons I have for so doing are two. First, the study of medicine we may liken to the distinguishing of objects seen through the haze and murk of a dense November fog : to those entering it from country sun-

shine topography seems lost, and the largest and plainest landmarks are seized as guides ; but when, owl-like, one's eyes become accustomed to the gloom, smaller objects attract the sight and interest. So do beginners in medicine seize first and gratefully the most evident signs and symptoms of disease : they love dogmatic views and the appearance of certainty ; but, as we grow older, we prefer to stray among more devious paths and take pleasure in noting petty details we failed to see at first. This is my case now : for, alas ! the years of my life are no longer few and the years are twice ten since I started on the serious study of medicine.

Again, beginners seek first knowledge (science) and for some time are, or should be, content therewith. But, as the days creep on, they grow weary of this abstract pursuit and cry *Cui bono*. This *Cui bono* cry will come quite apart from monetary desire or need. It is the natural outcome of the desire for knowledge, for this could only arise from the longing that is in us to do or say something useful, so that the years of our life be not wasted. Now art is the application of knowledge to effect a desired purpose, and so, as we grow older and more experienced, we begin to study the art rather than the science of medicine, the alleviation of pain rather than the knowledge of its causation and nature. Hence we take perhaps keenest interest in those phases of disease which are not fixed and enduring, but transitory and ever varying.

The causes of dyspnœa arise in the respiratory,

circulatory, or cerebral systems, or are due to mechanical obstruction of the heart or lungs.

**Respiratory Dyspnœa** is of three types :—

**Unconscious**, as in pneumonia ; **Conscious**, as in laryngitis and bronchitis ; **Voluntary**, as in dry pleurisy or peritonitis, fractured ribs, muscular rheumatism, or intercostal neuralgia. Over the first two forms the patient has no power, so long as their cause exists so will they, whether he will or no ; but voluntary dyspnœa is of his own creating, solely to lessen pain or help rib union ; there is no impairment of lung function.

The first thing to strike us in studying the symptoms of dyspnœa is this : sometimes it occurs without effort ; the patient lies on his back breathing with great and unconscious rapidity, each respiration being of small amplitude—*i.e.*, is shallow. The respiration of acute pneumonia is typical of this.

The reason is not far to seek. A certain number of air-sacs have been rendered impervious to air, have become solid ; it is evident there is no chance whatever of forcing air into these. Nature soon learns to accept the inevitable ; she at once forsakes the hopeless struggle, and does what she can with the rest of the lungs. Into this rest the air enters easily, but as its area is small the number of respirations must be increased. It is true that in acute pneumonia even, there are two epochs when dyspnœa may become one of conscious effort—*viz.*, at the beginning and as death comes. At the onset there is no exudation, but merely

hyperæmia ; the alveoli are not completely blocked, but their cavities are lessened by the distension of their walls with blood—the walls are thus made more rigid as well, and so are less easily stretched ; moreover, the terminal bronchiole's congested wall may partially stenose the entrance to the infundibulum. Here evidently then is some reason for struggle ; extra effort may allow the passage of more air through the narrowed bronchiole and more fully distend the stiff-walled alveoli ; further, the system has barely had time to learn the condition of affairs ; and, lastly, the heart is dumb-founded by the sudden shock, and conscious dyspnœa always comes to the aid of a failing right ventricle.

Towards the end of a fatal case failure of the right ventricle is the important feature, passive congestion and œdema resulting ; and conscious dyspnœa, laboured breathing, arises to help the feeble heart drive the water and blood through the tissues and clogged capillaries.

As the breathing changes from unconscious to conscious it usually changes also in other ways. Unconscious breathing is never deep and laboured ; for breathing to be deep, extra muscles under voluntary control are required, and the use of these, even though started by reflex means, draws the patient's attention to the act. Even in health this is so—*e.g.*, we have been reading a tale lazily cramped up in a chair for some time, quite unconscious of heart-beat and respiration, when suddenly a deep sigh recalls us to our unhygienic attitude.

Unconscious dyspnœa may last days, weeks, or months without intermission, just as we may have rapid

heart-beat so lasting. But conscious dyspnœa, if constant, is usually limited in duration, and if severe its continuance is a matter of days only : for these voluntary muscles have not the staying power of those engaged in perennial rhythmic action, nor has their nervous mechanism the same continuity of purpose, each impulse probably requiring a separate act of consciousness.

The inset of conscious dyspnœa in a case of acute lobar pneumonia is therefore a bad sign and one to be earnestly combated by cardiac stimulants and tonics, of which strychnine, administered intramuscularly in doses of five minims of the liquor every three or four hours, along with alcohol, is perhaps the best. Or the heart's labour may be lessened by venesection, treatment which is of doubtful value at a late stage of the disease, but the course *par excellence* to take at the onset.

One of the most marked examples of our next type—**Conscious Dyspnœa**—is that which occurs in the acute laryngitis of children. You all know the clinical picture—as pathetic a one as we ever have to look on. The constant unrest, now on that side, now on this, now supine or even prone the little body tosses. The head thrown back, mouth open, and nostrils eagerly working ; the arms in ceaseless movement with petulant weariness ; the diaphragmatic insuck of evil augury, constricting wasp-like the soft round trunk ; and, perhaps saddest of all, the piteous appeal of eyes telling of suffering and dread, along with the hurried moan begging ease from the pain of such labour. Can any other condition contrast more strongly with a similar child, in



the very next bed maybe, who is ill of acute pneumonia, lying quietly on its back ; the flushed cheek and rapid though easy breathing alone showing its sickness to a laic observer. But to the physician the cause of the difference is cogent. In laryngitis the air-sacs are healthy and patent, able and willing to oxidise the venous blood in their walls. But the block in the larynx hinders the ingress of the necessary oxygen. The blood hungers, the air-sacs yearn to satisfy it, they urgently appeal to the patient's consciousness ; this sets the accessory muscles of respiration in action, and conscious dyspnœa results. In unconscious dyspnœa no such appeal or yearning can arise, for through the blocked air-sacs no hungry blood can circulate.

In the membranous form of laryngitis—where the inflammation has so fatal a tendency to descend to the smallest bronchioles and even involve the air-sacs—a change comes over the dyspnœa. As death approaches this assumes more and more the unconscious type. Such alteration is due partly to a blunting of the voluntary respiratory centres from the unwonted, prolonged, and excessive strain to which they have been subjected, partly to exhaustion of the voluntary (accessory) muscles themselves, and partly to scattered patches of air-sacs becoming blocked, either from their involvement in the inflammation or from collapse. With this change in the dyspnœa comes too a change of countenance : the restless, turgid face grows calm and of an ashen hue—for the left ventricle is failing. Landois tells us that dyspnœa very rapidly raises the

peripheral tension, and hence gives the left heart far more work to do: also, this peripheral high tension constricts the coronaries as well as the other systemic arterioles, so that the heart muscle is imperfectly nourished. Thus ill-fed and subjected to excessive labour, the left ventricle, like any other living thing, soon has to cease its struggling: the turgid face goes, for the right ventricle has but little trouble in passing on the now too scanty blood supplied it by its feeble brother: the ashen colour comes, as the narrowed arterioles will only allow the worn-out ventricle to send a tiny stream along them. Thus is the act of dying one of calm peace, and death is ushered in with a *Requiescat*. Such, indeed, is the dread monarch's almost constant entrance: even half a century before our immortal ancestor wrote his "De Motu Cordis" Edmund Spenser summed up this fact in lines also immortal:

"Ease after toyle, port after stormie seas,  
Peace after warre, death after life, doe greatly please."

**Emphysema** produces respiratory dyspnœa: a dyspnœa which is usually a combination of the conscious and unconscious types.

Emphysematous lungs are unduly capacious: for each of their air-sacs to be as much distended as the air-sacs in a healthy lung during normal inspiration the cavity of the thorax must be made larger than it is in such inspiration—that is, inspiration must be aided by the use of the voluntary muscles, and hence the

breathing will become conscious. You may object to this that an increase in the rate of the inspirations would affect the same interchange of gases and be far easier to the patient. To this my answer is obvious: in health—that is, naturally—the air-sacs are accustomed to a certain degree of distension, a condition which long inheritance has no doubt made most suitable for the due nutrition and functions of the lungs: this degree, therefore, they will always seek to attain. Thus it is that in emphysema, whenever there is any insufficient interchange of gases, the patient endeavours to obtain the sufficiency in the most natural way—viz., by increasing the amplitude of the respiratory movements.

Further, in emphysema, some air-sacs are destroyed and their function gone—just as occurs temporarily in acute pneumonia—and hence, as in pneumonia, there is a quickening of the respiratory act—an unconscious dyspnœa—which in this case is unfortunately permanent.

You will object, again, that should these be the true explanations, then should emphysematous subjects be constantly suffering this double form of dyspnœa. I answer you that some do thus suffer without ceasing. The majority do not, because man is blessed with an immense respiratory reserve, the full amount of which is only called into action at times of extreme stress, and that man must be in a sore strait whose emphysema is so great that he has to call upon his accessory muscles even at times when he sits quietly at rest.



Another cause of respiratory dyspnœa is **Pulmonary Inadequacy**. You are all cognisant of the phrase renal inadequacy : there is similarly a congenital feebleness of function, or anatomical smallness, of the organs of respiration. So strikingly is this the case in some instances that the question of congenital emphysema has been mooted. But there is no physical sign whatever of emphysema : two or three subjects in whom I have observed it have been unusually healthy and vigorous, not only as to their muscular system, but in possessing essentially a strong constitution. They can perform a very large amount of light physical labour and of mental strain, but any greater exertion, such as walking uphill, initiates dyspnœa, which easily and quickly goes on ceasing the labour. This dyspnœa is in no way cardiac, for, even during its continuance, the pulse is firm, steady, and of but moderate rapidity.

Cognate to this subject of respiratory dyspnœa are the conditions of **Collapse and Œdema**. Collapse is due to the more or less complete obstruction of a bronchiole by a plug of mucus or foreign particle. The air remaining in the shut-off lobules rapidly loses its oxygen, becoming rich in carbonic acid instead ; for the alveoli are able to extract almost the whole of the oxygen in the air supplied them. The carbonic acid is very speedily reabsorbed by the alveolar walls, being soluble in its own bulk of water, and the nitrogen more slowly follows suit. If there be feeble circulation, with some anasarca of the lung tissue, and the respiration be also feeble—conditions which obtain in old age,

especially when the patient is kept supine so that the contraction of the diaphragm is not aided by gravity—then water not seldom takes the place of the absorbed gas, ascites of the air-sacs resulting. When the collapse or alveolar ascites is complete, the respiration will undergo an unconscious quickening in proportion to the amount of lung involved ; unless, indeed, this be so small and the lungs be otherwise so healthy that the pulmonary reserve is not seriously encroached upon ; but unfortunately in these instances the lungs are nearly always considerably affected in other ways. So long as the annihilation of the cavities of the air-sacs be not complete, so long will some conscious dyspnœa exist along with unconscious, the proportionate amount of each type depending on the degree of patency of the involved air-sacs.

**Circulatory Dyspnœa** is a symptom of far more complicated origin than that due to pulmonary defect. It may arise from malfunction of the left or right heart, of the systemic or pulmonary arterial system, or from an impure or impoverished state of the blood.

Usually it is caused by a combination of one or more of these. If the right heart or pulmonary system, or both, be involved, the dyspnœa is nearly always of the laboured conscious type, whether the left heart be also at fault or no. Perhaps the chief differentiating characteristic of this form of circulatory dyspnœa is its marked dependence upon physical exertion. Unless of great severity it does not exist when the patient is at rest, but very little physical exertion suffices to bring it

into prominence ; it is the dyspnœa of exertion *par excellence*. I would not have you understand by this that respiratory dyspnœa is not exacerbated by exertion, but on respiratory dyspnœa physical strain has far less influence, and, moreover, such dyspnœa is nearly always present to some extent during rest.

**Air Hunger**—the desire for a sigh—is a type of dyspnœa quite *sui generis*, and is pathognomonic of defect in the left heart or systemic arteries ; not necessarily organic defect : functional spasm or weakness is equally effective in its production. This is no more dependent upon exertion than is respiratory dyspnœa, thus again differing from dyspnœa of the right heart or pulmonary arteries. Often it is not recognised as dyspnœa by those who suffer from it, but is spoken of as a constricting band around the chest, as a weight or sense of oppression beneath the sternum, or as a longing for more air, which longing cannot be satisfied by any voluntary increase of respiration. Of this air hunger aortic regurgitation with failing compensation is perhaps the best known example. In this condition the respiratory centre has a too small supply of blood ; moreover, this smallness of supply has the further disadvantage of being intermittent. All the constituents necessary for the centre's well-being are thus deficient ; it languishes therefore, but its languishment for oxygen is the most imperative and urgent, for without oxygen it is not only unable to build up its cells with fresh nourishment, but cannot even get rid of the excrement produced by their life. Hence this life, the centre's function, is continued

under difficulty, clogged as are its wheels with dirt. In its trouble it calls aloud for more oxygen, but soon perceives—one or two of its messages to the lungs and right heart are sufficient—that the air-sacs are patent, no tubes are obstructed, and that the right heart sends on its blood easily and well without undue labour.

These things being so it cannot initiate conscious or unconscious dyspnœa and can only express its helpless condition by an appeal through the vagus for extra effort on the part of the left heart ; finally, the inability of this to respond to the appeal produces the discomfort or pain of air hunger.

Air hunger is a striking symptom in affections of the myocardium, whether these be degenerative, inflammatory, or due to pericardial adhesion. Here it is evident both sides of the heart are equally open to the evil influence : yet it is air hunger and not laboured breathing which results. The reason for this is plain. Any flaw in the tissues tells most in the organ working at the higher pressure. Moreover, if the quality of the material of which both are made be equal, it is that belonging to the engine working at the higher pressure which will first begin to give way. You will say, were this so then would the left ventricle always be the first to fail, even in those fortunate beings where neither ventricle is subjected to any special strain. But this does not necessarily follow by any means. During systole the blood-vessels of the left ventricle are subjected to a greater pressure than those of the right : they will therefore be the more completely emptied, and there-



fore during diastole will receive a greater amount of arterial blood. In other words, the left ventricle is better fed than the right, and we all know that the better a horse is fed the more able he is to withstand the strain of prolonged labour. Yet, notwithstanding this, you will find that death comes from failure of the left oftener than of the right ventricle, even in those cases where we can find post mortem no disease nor reason for special strain of either. Hence it is that, clinically, an affection of the myocardium is almost exclusively evidenced by failure of the left ventricle, and that air hunger, not laboured breathing, is the peculiar cry of the distressed myocardium.\*

Sudden access of contraction of the peripheral arterioles will not infrequently originate air hunger even where no cardiac or organic arterial defect can be discovered: such will occur from evil metabolism after a full meal or on sudden exposure to low temperature. The condition induced is essentially one of feeble blood-supply to the respiratory centre owing to the heart being unable to pump up the normal supply against the greatly increased resistance.

**The Dyspnœa of Debility** is a circulatory dyspnœa of more complicated nature. Here the root of the evil

\* In my experience the septum ventriculorum is much less often diseased than the rest of the wall of the left ventricle, and this I believe is due to its being subjected to a greater pressure and its vessels therefore more completely emptied during systole. They are doubly compressed between the tension of the blood in both ventricles, whereas the external layers of the rest of the left ventricular wall experience a negative pressure.

is a deficiency in the powers of digestion or assimilation, or both. A given quantity of blood hence contains less nutritive material than normal ; every organ gets below par ; the brain and heart, as the most highly organised and most constant in action, suffer most ; prolonged labour by either becomes impossible ; both right and left heart suffer, hence there is a tendency to both air hunger and laboured breathing. The “desire for a sigh” is felt after a continuance of brain effort or moderate physical exertion ; but, as I have explained in the Ingleby Lectures (Lecture II.), any sudden strain, physical or mental, falls most on the right ventricle, and hence initiates conscious dyspnœa. Should the impure blood in debility produce vasomotor spasm, the work of the heart will be both intensified and its nutrition (owing to contraction of the coronary arteries) lessened ; and now will be added to the previous dyspnœa a continuing sense of præcordial oppression and tenderness, the heart often feeling like a great heavy tender tooth, and the præcordium itself becoming exquisitely tender to palpation.

**True Spasmodic Asthma**—by which I mean an asthma the first attack of which occurs where we can find no lesion of lung or heart—is the most interesting of all dyspnœas. It does not follow that no such lesion exists ; but if it does it must be of so small a nature as to be quite unequal to produce an attack in ordinary people. True spasmodic asthma is therefore a reflex spasm started by an irritation which is too feeble to produce any such result in a normal condition. Many

men have nasal polypi, yet never asthma; many suffer from evil metabolism (migraine, *e.g.*), yet have no asthma; many have bronchitis, yet never asthma. Hence a peculiar predisposition is evidently necessary. It may be due to an excess of irritability in any portion of the reflex arc, and its occurrence being almost confined to people of high nervous organisation would point to a *general* excess of nervous irritability; but, on the other hand, the great variety of afferent impulses which consistently produces this, and yet no other form of nervous explosion, points still more strongly to instability of the central and efferent portions of the arc.

However excited, the immediate mechanism by which the spasm arises is triple—viz.: (1) a sudden swelling of the bronchial mucous membrane; (2) a sudden contraction of the bronchial muscle; (3) a vasomotor contraction of the pulmonary arterioles. In any given case one or other of these is the most prominent, but as a rule all three are at work in greater or less degree. Direct ocular proof that any one of these three exists during a paroxysm is, in the nature of things, impossible; but the indirect arguments in their favour are extremely strong. Regarding the first, we know that in an allied membrane, the skin, direct irritation, such as the sting of a nettle or insect-bite, is capable of producing it in a minute or so,\* while

\* It is interesting to note that Sir William Gull wrote a short paper to show that such urticaria was, in large measure at any rate, due to a spasm of the cutaneous muscle fibres.—Works of Sir W. Gull, vol. i., New Sydenham Society, vol. cxlvii. p. 566.

products of evil metabolism or those resulting from injurious food (shell-fish) induce a generalised swelling (nettle-rash). Moreover, it is well known that some people are far more sensitive to these influences than others. Again, a still more nearly allied membrane, the bucco-pharyngeal, is seen to undergo transitory swelling of the same nature, and I suspect that some cases of acute glossitis are to be thus explained. Finally, irritants of similar or identical nature with those which cause asthma, often set up in a few minutes, when inhaled through the nares, an acute and severe swelling of the nasal mucous membrane, which we may almost call a portion of the bronchial tract, so closely is it related to this in function. But hay asthma is perhaps the most convincing proof of the existence of an acute transitory swelling of the bronchial mucous membrane in bronchitic asthma. Hay asthma, we now know, thanks to Dr. Blackley's able experiments, to be usually due to irritation of the nasal mucous membrane by the pollen grains of some grasses. An hour or so, or even less, after their inhalation acute swelling of the membrane is seen to occur, followed by paroxysmal sneezing and profuse discharge, the whole cycle of events not occupying more than a couple of hours, and being followed by complete health, until the numerous repetitions initiate changes in the membrane which cause a constant sense of discomfort. It is evident this nasal paroxysm is the result of reflex action ; its intense and transitory nature and its generalisation throughout the whole nasal lining stand out in too great contrast to an



inflammation arising from a merely local irritation, to enable us to put it down to this cause. Now sometimes the nasal asthma is accompanied by dyspnœa ; the nasal asthma becomes naso-bronchial asthma. Surely it is only reasonable to suppose that the pollen grains have directly irritated the bronchial membrane, or that their irritation of the nasal membrane was so intense that the stimulation set up was not confined to the nasal centre, but overflowed into its near neighbour the bronchial centre (if, indeed, this be not another part of the same centre). Whichever supposition we choose—and probably both are right—we can only conclude that the reflex influence causes a disturbance in the bronchial mucous membrane similar to that which occurs in the nasal. Moreover, it is hard to understand the numerous râles and profuse expectoration which usher in the later stages of many attacks of asthma unless we admit a disturbance in the mucous membrane.

At present the theory that asthma is due to a spasmodic contraction of the circular unstriated muscle of the bronchial tubes is the one most generally held. Michael Foster tells us that the bronchi and larger bronchia are distinctly muscular tubes, and that after death their muscle fibres, contracting, throw the mucous membrane into longitudinal folds : also, that in the smaller, non-cartilaginous bronchia there is still a considerable number of these fibres ; while Klein, in his “Atlas of Histology,” says that “the wall of the alveolar ducts and infundibula are chiefly made up of unstriated muscle cells running in a circular manner.”

Berkart, in his learned monograph on asthma, quotes Rindfleisch as saying that towards the infundibula the muscle layer forms a kind of sphincter. We must admit, then, that on *a priori* grounds at any rate it is almost certain that so strongly developed a contractile constituent as this must have a great deal to do with any sudden, transitory, and widely distributed obstruction of the bronchial tubes. Judging from what we know of the arterial muscle layer, one of the chief duties of this bronchial muscle should be to prevent the entrance of deleterious air into the air-sacs by obstructing the lumen of the tubes. It has been proved experimentally by C. J. B. Williams, Paul Bert, Volckmann, and others that stimulation of the vagi produces contraction of the bronchial tubes, and that this contraction is the greater the more collapsed the lung. This is just what we should expect were the contraction due to the bronchial muscle, for the more collapsed the lung the less would be the force of its elastic tension, which must act as a dilator of the bronchi. This does away with Berkart's objection to the theory of muscular spasm—viz., that the dyspnœa of asthma is mainly expiratory, whereas if it were caused by muscular spasm it would be equally inspiratory. Besides, we must remember Klein's statement that "bands of unstriated muscle cells are absent from the greater part of the alveolar wall": thus the muscular spasm would not compress the air-sacs but only the tubes by which the air escaped from them. Lastly, there is little doubt that the muscles of inspiration are more powerful

than those of expiration, for, to take one instance only, the diaphragm, the most powerful of all the respiratory muscles, is only in action during the inspiratory period. Further, the contraction of this muscle layer would increase the obstructive action of the mucous membrane: for Foster tells us that "after death, owing to contraction of the circular muscular fibres, the mucous membrane, like the internal coat of an artery in the same circumstances, is thrown into longitudinal folds."

I want, however, more particularly to draw your attention to the third mechanism I have mentioned, inasmuch as it is one to which I can find no reference in relation to asthma. As yet we know but little of the pulmonary vasomotor system. Yet of these two facts we are cognisant; first, that the pulmonary arteries possess a circular muscle layer, though this is neither so well developed nor so continuous as that of the systemic arteries; and secondly, that vasomotor fibres from the lungs reach the medulla through the upper intercostal nerves (2nd to 6th, both inclusive): in other words the vasomotor centre has direct vasomotor communication with the pulmonary arterioles.\* We also know, from examination of the pulmonary second sound, that the tension in the pulmonary arteries is constantly varying, not only from day to day, but from hour to hour, and this variation can arise quite independently of any cardiac lesion. There is every reason, therefore, to conclude that the pulmonary vaso-

\* Bradford and Dean, *Journal of Physiology*, 1894

motor system is liable to attacks of high tension in common with that of its greater brother.\*

There seems little doubt then, that a sudden transitory anæmia of the pulmonary arteries can occur. If it did occur, what would the symptoms be? Embolism of the pulmonary artery has not seldom been mistaken for an attack of asthma. Berkart quotes such a case recorded by Virchow : a man of seventy-six caught cold, bronchitis arose, and this was soon complicated with severe asthmatic seizures, in one of which he dies ; the diagnosis made was bronchitis with asthma, but post mortem Virchow found that the second divisions of the pulmonary artery contained several plugs of different ages. Embolism of the pulmonary artery, therefore, produces attacks closely resembling asthma ; we should expect sudden peripheral tension to do the same, for the effect of both it and embolism is to cause a sudden pulmonary anæmia.

Some have supposed that a prolonged tetanic spasm of the diaphragm is the cause of asthma, since the chest remains so continuously distended. Vasomotor spasm would explain this, whereas bronchial obstruction does not. If we suppose vasomotor contraction alone to be the cause of an asthmatic attack, there will then be no

\* The pulmonary high tension occurring in mitral disease is of course not due to peripheral spasm but to back pressure from the left heart. The accented second sound in anæmia points to peripheral high tension, but I am by no means sure that it is not due to the dilated and distorted condition of the pulmonary artery, though the existence of such tension may be a factor in the production of anæmic dyspnœa.



hindrance to the entrance of air into the air-sacs, yet but little blood will be oxygenated, as the contracted vessels will prevent its due contact with the air. So long as the pulmonary arterioles remain contracted there thus appears no way of getting the blood sufficiently oxidised. But though due oxidation of blood cannot take place yet there is a way by which the respiratory centre can be appeased, temporarily at any rate. Mere distension of the lungs with a neutral gas, as by repeated inflations of hydrogen, will induce apnœa—*i.e.*, appease the centre.\* It is only natural to suppose, therefore, that the lungs, in the dire strait into which vasomotor contraction has thrown them, will perpetrate this pious fraud upon the centre and, by a series of forceful inspirations, endeavour to ward off its dyspnœic irritability, thus producing a condition closely allied to tetanus of the diaphragm.

No similar reason for great and continuous distension of the lungs exists in spasmodic narrowing of the bronchi alone. Indeed, it does not occur in the dyspnœa of membranous croup, which post-mortem evidence shows to be due to widespread bronchial obstruction.

We should not expect it: there is no need for an abnormal amount of air to be inhaled, for the air-sacs and vessels are only too ready to make good use of what air is afforded them: the interchange of gases is rapidly effected, and the expiratory forces at once petitioned to remove the used-up air.

Some asthmas are very intimately connected with

\* Foster's "Physiology," 5th ed., p. 604.

food. I know a medical man who always had an attack if he ever took supper, though not otherwise, during a winter when he suffered much from bronchitis. Hyde Salter says, "Errors in diet are a very fruitful source of asthma." But he explains their action by the intimate nervous relation between stomach and lung—the food in the stomach reflexly exciting the lungs—yet a case he uses as an example does not point to the presence of food in the stomach as the exciting cause, for the patient woke up in the early morning with asthma if he took any food later than 2 P.M., and one can hardly suppose that the food would so long remain in the stomach. Indeed, the paroxysms seldom occur less than four to six hours after the evening meal—that is, after the food has left the stomach—whereas, if it were from gastric irritation, surely two to three hours after a meal—the time, that is, when dyspeptic breathlessness and palpitation most frequently arise—would be their favourite moment. Besides, they rarely occur till after sleep has overtaken the patient; and if he should determine not to retire to bed till after the usual hour of attack he not seldom escapes altogether. Now during sleep gastric irritation is less perceived: the gastric, like most other sensory nerves, are asleep as well as the cerebrum: hence, if the waking state be maintained, one would expect the paroxysms to be severer than if sleep were gained. But during sleep the oxidation of the blood, both from decrease in respiration and from slowing of the pulse, is less thoroughly performed: therefore in sleep the arterioles

would tend to become still more strongly contracted from increased toxicity of blood: hence sleep would tend to induce a paroxysm if this were caused by contraction of the pulmonary arterioles. Such attacks as these, then, are by far most easily explained by evil metabolism producing vasomotor contraction of the pulmonary arterioles. They do not arise till almost the whole of gastro-intestinal digestion is over—that is, not till metabolism is in full force—and, if evilly performed, is allowing improper material to enter the circulation. It is quite a common experience to note the radial artery grow small and hard some three or four hours after an unwholesome meal, and it would be strange if the pulmonary system were not affected in some similar fashion. By this vasomotor contraction you must understand something quite different to the “hæmic dyspnœa” of Walshe, for in Walshe’s dyspnœa there was no lessening of the freedom of respiratory movements, the air entered and was expired in even larger quantity than usual: it resembled the breathlessness of chlorosis. But in speaking of true spasmodic asthma it is well always to connote a great diminution of respiratory movement, and I have previously explained such diminution would be the natural consequence of spasmodic contraction of the pulmonary arterioles.

All asthmatic paroxysms, even those occurring in extensive organic disease of lung or heart, must be due, immediately, to spasm of some kind. If a man is labouring with the dyspnœa of advanced emphysema, a very small spasm, or sudden additional obstruction to

the gaseous exchange, may be sufficient to induce the paroxysm. Still such spasm, however small, must occur ; therefore the immediate cause of all asthmas, bronchitic, cardiac, renal, gouty, nasal, or what not, is one or more of the three mechanisms of which I have spoken.

**The Management of Dyspnœa** I can best treat of by detailing to you some cases which have been under my own care.

Mrs. S., 39, who kept a small shop, I saw in consultation with Dr. Vince. She had suffered great anxiety, having to keep her husband as well as herself : for the last eighteen months she had been too feeble to attend to the shop, and this had further increased her worry. Attacks of præcordial pain had, during this period, been frequent. For three weeks she had been considerably worse, and the anginal pain constant, though subject to exacerbation. Œdema of the legs and abdominal wall also existed with congestion of the bases of both lungs.

The heart's apex beat was in the fifth space outside the nipple line : on the right the dulness reached to the right edge of the sternum and upward to the third cartilage (hence an enlargement of the right as well as the left ventricle). The impulse was short, sharp, and diffused. The sounds were cantering, and there was a well-marked mitral systolic murmur which I believe was due, not to endocarditis, but to dilatation of the valve ring. The pulse was very small, irregular, and not easy to feel ; rate about 100. She had orthopnœa and insomnia. The anginal attacks were worse at night, but never apparently very agonising.

Here was evidently a grave affection of the myocardium, so grave, indeed, that the right ventricle showed symptoms equally ominous with those of the



left—viz., pulmonary congestion and anasarca. These probably arose only with the exacerbation of the last three weeks, for I have pointed out to you how much longer the right ventricle fights against causes inducing degeneration of its walls than does the left. What was the cause of this myocardial degeneration? I think in this instance the train of causation was worry, evil metabolism, impure blood, vasomotor contraction of systemic arterioles, including the coronaries, inability of ill-fed heart to fight against its extra labour. When I saw her the heart was too feeble to produce high tension, and the pulse was as I have stated above. Still, that there was potential high tension was shown by the marked action of nitro-glycerine, for after a dose of  $m \frac{1}{20}$  the pulse doubled in size, and at the same time greatly modified and shortened an anginal spasm if this were present. Indeed, this case was remarkable for the great control nitro-glycerine had over the dyspnœa and pain. The patient kept a six-ounce bottle of a  $\frac{1}{6}$  per cent. solution by her side, and whenever she felt a spasm approaching drank a few drops direct from the bottle, drinking the whole six ounces in a week—*i.e.*, nearly five grains of pure nitro-glycerine.

But even this largeness of dose I find has been greatly exceeded. Dr. Murrell had a patient, a man of thirty-three, suffering from aortic regurgitation, who took the drug almost without interruption from June 1879 to January 1882, for 126 consecutive days of this period taking one ounce of the 1 per cent. solution daily, and during two whole years a daily average dose of

half an ounce. All this period he worked on the average four days weekly, for several weeks working sixty-seven hours. He was a printer, and he took the drug to combat attacks of angina, which it did with wonderful success. Here then are two examples of the excellent effect of a vasomotor dilator in feebleness of the left ventricle, accompanied by contraction of the peripheral vessels.

Mr. G. H., 61, a twin, consulted me concerning loss of strength and breath, with a sleep-disturbing cough. He was a coach harness-plater by trade, a steady man and a hard worker. His father died of apoplexy at 64, his mother of fatty heart at 58, and one sister of a stroke at 60. Since boyhood he had suffered from indigestion and from palpitation on exertion. The indigestion was at times severe; fifteen years ago he was laid up for eight weeks with it. For the past eight years he has had winter cough, but this, till lately, has not affected his breath. He has no paroxysms of dyspnœa, but breathing is a constant labour to him. The expectoration is fairly free, but he is usually conscious of wheezing. On examination I found the chest very barrel-shaped, the liver dulness beginning at the seventh cartilage. There were patches of deficient P.N. in several places, and over them dry crackling was heard. This was especially the case at the bases of the lungs. The liver was felt two inches below the ribs. Heart: A.B. fifth space and n.l. v.D. lower edge of third cart., R.D. one inch to R. of sternum: impulse thrusting and first sound is thumping; the second sound is accented and rough all over the præcordium; sometimes the beats are coupled, a small one following a large, the action is always very irregular, no murmur heard. The jugulars fill up just before the first sound, and remain full up to the second; on lying down the jugulars are continuously

distended, but some transverse veins still empty after the second sound. The pulse is very small, and the brachial artery is thickened.

In this patient there was evidently an inherited weakness of the vascular system shown by the family history, the life-long palpitation, and the indigestion which was probably vascular in origin. On the top of this inherited weakness he contracted a bronchial catarrh; the mucous membrane never fully recovered, so that easy recurrence came with each succeeding winter. He sustained this additional mischance without any additional dyspnœa for seven years, and then his right heart began to fail, and laboured breathing at once ensued. Examination showed that he had damaged lungs with bronchitis, both sides of the heart enlarged, and a failing right ventricle, as shown by the distended veins and swollen liver. The lungs were evidently at the bottom of his present dyspnœa, and I gave him this mixture :

R Pot. iodidi,	gr. 3.
Extr. stramonii,	gr. $\frac{1}{4}$ .
Extr. glycyrrhizæ,	gr. 2.
Ætheris sulphurici,	℥ 5.
Liq. arsenicalis,	℥ 2.
Aquam, ad	℥ j.

To be taken five times a day.

In a month I saw him again. He now slept pretty well. His cough had ceased to trouble him, and his breathing was much easier. All wheezing and rhonchus had gone. The R.D. of the heart had returned as far as the R. edge of the sternum and the V.D. to the upper edge of the fourth cartilage. The expectoration was now very slight. The pulse, though still irregular, was less so, but the veins still showed the same over-distension.

Mr. F. I., age 60, a carpenter and undertaker, was brought to me by Mr. S. Trout. The family and previous histories were good. Nine months before his visit to me he was acutely seized with severe dyspnœa the nature of which we could not determine, as he was then not under Mr. Trout's care. It confined him to bed for a fortnight and laid him aside for another month. He returned to work, though far from well. In three weeks went to Hastings for change of air : came home no better, but again went to work. Three months later went to Yarmouth, where he had two or three severe attacks of nocturnal dyspnœa. Came home and to work once more. Improved somewhat. Again went for a change to Aberystwith : returned worse, and has since been getting weaker and able to accomplish but little work.

On examination he was a powerful, well-built man with a dusky countenance, evidently weak and dyspnœic. His p. was 118, sudden, rather small and dicrotous. Vessel much thickened. Heart : A.B. diffused in sixth space, one inch outside the n.l. ; R.D. 2 inches to R. of sternum and v.D. at third cartilage. The apical sounds were cantering, and there was a soft, barely heard, systolic murmur, localised to the position of the A.B. Urine 1025, thick cloud of albumen, much urates, no sugar. There were a few bronchial râles at the bases of the lungs. The liver was not enlarged. There was slight œdema of the legs, and he had sweated much at night during the last three weeks.

He was ordered to confine himself to one floor of his house, to wear a belladonna plaister, to take  $\mathfrak{M}_{\frac{1}{100}}$  of glonoin for any access of dyspnœa, and a grain each of powdered digitalis, fresh squill, and blue pill as a pill every night ; with this medicine :

℞ Tr. nuc. vom.,  
 Tr. digitalis, āā,      ℥ 15.  
 Sp. Ammon. Arom.,    ℥ 30.  
 Aquam, ad              ̄j.

To be taken three times a day.



He came again in a month with his heart much smaller. A.B. fifth space 1 inch without n.l., R.D.  $\frac{1}{2}$  inch to right of sternum, and v.D. middle of third space. The A.B. was localised and the impulse of good quality, though to the stethoscope there was still a little tendency to a cantering sound, and a slight systolic murmur still existed. The second aortic sound was musically accented. The P. 82, of high tension, but beat full and sustained. The lungs were clear, and the urine 1010, clear and pale, a faint cloud of albumen, and "pretty free" in quantity.

He felt better and stronger: his dyspnœa had gone, except a little after a good dinner on one occasion; his face was bright and clear; œdema of legs gone. The digitalis was omitted from the medicine after the first week because of nausea, and the pill he only took for ten days.

The history here is obscure, but examination of his radial told me he was suffering from vascular degeneration in excess of what is normal at his age. With this enduring weakness, he contracted severe acute illness nine months ago, which was subjectively chiefly characterised by dyspnœa; was therefore very likely bronchopneumonia, perhaps influenzal in type. The musculature of the heart never recovered from the shock. The left ventricle was chiefly affected, but the mitral leakage, as indicated by the mitral murmur, at last upset the function of the not strong right ventricle; dilatation of this also ensued, as shown by the area of cardiac dulness, and the lungs were beginning to suffer. Rest was evidently the great desideratum here; to it I added nuxvomica to improve the myocardial nutrition, and digitalis to regulate and slow the heart's rapid irregularity.

The mercury and squill were given to increase the action of the liver and kidneys, and so do away with the lithiasis, the albuminuria and the œdema.

Mr. A. R., an organist, 55, consulted me for a shortness of breath from which he had suffered for four years. For five months during the preceding winter this was accompanied by an annoying whistling sound in the lower part of his chest, but this noise had ceased during the last four months. Careful examination failed to detect anything wrong in his lungs and heart, though his pulse was of high tension and short duration, and his temporal arteries prominent and tortuous. His previous history was quite good, as was also that of his family, with the exception of his father, who died suddenly at 51. The edge of the liver was felt hard and thin, as if cirrhotic, half-way to the umbilicus, though the upper margin of its dulness was normal. This condition of the liver I was and am unable to account for. There was a faint haze of albumen in the urine, but no sugar. He also complained of a feeling of fulness in the nose, as if no amount of blowing would clear its passages. On examination I found a large polypus completely blocking the left posterior nares. The naso-pharynx and pharynx exhibited granulations of considerable chronicity, while the superior surface of the larynx showed a subacute inflammation, and both cords were pink.

The polypus at once accounted for the dyspnœa. It had set up considerable local irritation in the naso-laryngo-pharyngeal cavity, and by the aid of this mischief of its own producing it had caused spasm of the bronchial muscle, more or less permanent in character, but of ever-varying degree (for the amount of the dyspnœa was by no means constant). When one

remembers the almost constant (though ever-varying in degree) spasm of the vasomotor fibres which arises from the toxic state of the blood in cases of granular kidney, this permanent reflex result of a continuing irritating cause will not be hard of credence. I at once sent him to Mr. Marsh to have his polypus removed. This was done with great and lasting benefit to his dyspnœa, though the degenerated condition of the nasal membrane still gives him considerable annoyance.

Mr. T. R., 52, draper, of nervous temperament, is another similar but more complicated case. About three times a week for the last three years he has waked early in the morning with his nose blocked : after sneezing and free discharge for one or two hours this passes off, but the same condition usually returns towards evening. For a fortnight there has been an exacerbation, every night about 1 A.M. he has waked with a suffocative cough, the distress lasting some two hours.

The heart's apex was in the n.l. and lower edge of the fifth space, the R.D. normal, and the V.D. depressed to the lower border of the fourth cartilage. There were systolic murmurs localised to the apex and the junction with the sternum of the second, third, and fourth right cartilages ; murmurs, I believe, due to mitral and tricuspid regurgitation from dilatation of the valve rings. The lungs were emphysematous, while crackles and wheezes were heard at both bases. P. 82, rather hard, short, small ; vessel thickened.

The urine contained a haze of albumen but no sugar.

This case I looked upon as one of bronchial spasm arising from bronchitis and emphysema, and made the diagnosis of bronchitic asthma, made worse by a fresh

outburst of catarrh. I therefore gave him this prescription :

R Pot. iodidi,	gr. 3.
Extr. stramonii,	gr. $\frac{1}{4}$ .
Extr. glycyrrhizæ,	gr. 2.
Ætheris sulphurici,	℥ $\frac{5}{8}$ .
Aquam, ad	℥j.

To be taken at 2, 4, 6, 8, and 10 P.M.

He came again in three weeks, stating that he had lost his suffocative cough, and that his nose had been much clearer, though he still suffered from oppressive shortness of breath. Both cardiac murmurs had gone, thus tending to confirm my idea that they were functional. The v.d. had now sunk to the fifth space, showing that the heart was previously enlarged and also that emphysema existed. The cardiac action was much improved. The lungs appeared to be only slightly drier, but the amount of moisture in a bronchitic lung, or perhaps, I should say, the evidence of it, is a very transient quantity. Yet as he still had dyspnœa which was increased at night, and as he still complained of stuffiness of his nose, I examined his posterior nares and found each blocked by polypus ; there were also some large flabby granulations on the posterior wall of the pharynx. I thereupon sent him to Mr. Haslam, who removed both polypi. I saw him again a fortnight later, during which time he had continued with the medicine thrice daily. He had, since the removal of the polypi, suffered no dyspnœa at night, though he still woke at 1.30 (probably from the habit his asthma had forced him to acquire), and on waking always found a



quantity of loose phlegm at the back of his throat. On examining his posterior nares I found that though the polypi I previously saw were gone yet another existed about midway in the right nostril, and the mucous membrane was swollen.

This is a good instance of the necessity of ever making a thorough examination of the body, however satisfactory may seem to you the condition of any portion as an explanation of all the symptoms. Here I felt convinced that the asthmatic attacks were fully explained by the state of the lungs and heart and thought the somewhat vexing examination of the nasopharynx unnecessary; but the progress of the case has left no doubt on my mind that the polypi were the exciting cause of the spasmodic attacks, though the vascular and emphysematous degenerations no doubt largely predisposed the patient to their irritation, and magnified into a large reflex spasm what would otherwise have merely been a local disturbance.\*

Mr. W. J. N. is a third case of somewhat similar though more complicated nature. He was sent to me by Mr. T. Law Webb of Ironbridge some four years ago, and I have seen him at intervals since. He was then 62, tall, spare, shoulders rounded and high, but well built otherwise. His only brother died of consumption at 24, and his father suffered from bronchitic asthma though he lived to 80. Since manhood he

\* I have since seen this patient in an attack of acute bronchitis, and I then gathered that the attacks of spasmodic dyspnœa had not quite ceased, but had probably improved not much more than in proportion to the general improvement; leaving the etiological action of the polypi a small and uncertain factor.

had always experienced great susceptibility to catarrh and cold. During the last ten years he would have two or three attacks of asthma, between 2 and 3 A.M., on successive nights, after which would come an interval of freedom for as many months. For four years he has been subjected to rather severe attacks of bronchitis. Meanwhile the acute attacks of asthma have gradually left a permanent dyspnœa which is liable to exacerbations. During this period, however, he has taken life more easily and this may account for his freedom from spasmodic asthma, whilst he holds that his general pulmonary condition has deteriorated.

On examination I found marked and well-nigh universal evidence of emphysema. There was no cardiac dulness and the hepatic note was not heard till the seventh rib. The heart's apex beat in the fifth space and n.l. The liver was I think enlarged, as it reached down to the umbilicus. A long wheezing expiration followed a short inspiration, and the heart's action was laboured with a heaving impulse.

The urine was healthy.

On examining the nares and pharynx considerable thickening of the lining membrane of an uneven granular type was noted, and six months previous to his visit to me Mr. Barling had removed two nasal polypi.

Here the condition was chronic and in great degree irremediable. Prolonged action of catarrh had caused degeneration of the lining membrane of nose, pharynx, and bronchi with resulting alveolar dilatation. This in its turn had enlarged and weakened the heart; the weakness being shown by the great variations in the pulse during the movements of respiration, and also by the enlarged liver. The position of the apex best showed that the enlargement of the heart was chiefly of

the right ventricle and the character of the dyspnœa was consistent with this. The expectoration was typical of the condition ; two or three ounces in the 24 hours of clear and very viscid mucus, brought up in small quantities after much spasmodic coughing.

There were three indications for treatment : first, to relieve the considerable amount of spasm which existed, and for this I gave stramonium : second, to strengthen the heart, for which purpose strophanthus appeared to me the most suitable drug : thirdly, and perhaps chiefly, one had to endeavour to improve the flabby, chronically inflamed mucous membrane, and to this end I know of no adjuvant so valuable as iodide of potassium. I also advised him in several details to take life still more easily. In a fortnight his statement was : "Much benefited ; but little difficulty in walking quietly upstairs ; but little expectoration ; only slight dyspnœa ; have been more careful in my life." Since then his condition has been chequered, but on the whole on a better level ; any temporary catarrh or dyspnœic exacerbation has always been greatly alleviated by resource to the above medicine or some slight modification of it. Twice since, he has again had polypi removed from his nostrils because they so blocked up the nares as to be a source of great discomfort and constant naso-pharyngeal catarrh. But though removal always eased his condition, yet I never made out that the polypi either produced attacks of spasmodic dyspnœa or that these, or the permanent dyspnœa, were distinctly relieved by their removal. We must, however, remember here

that, no matter how skilfully and thoroughly the polypi were removed, their removal could not make good the damaged and rugose mucous membrane, so that the nasal condition ever remained a source of constant reflex irritation.

This gentleman suffered much from a very common pain in these cases ; I refer to a dragging tender sense in the epigastrium which I think was a sort of diaphragmatic neuralgia from overstrain. The diaphragm had to do its work in a constant condition of contraction owing to the emphysema ; it was never completely relaxed, and in the patient under discussion it was further hampered by an enlarged liver.

I have since heard from Mr. Webb (October 26, 1894), as follows :—"The eucalyptus oil capsules (m 5 in each, to begin with 4, and increase up to 12 daily, if necessary—prescribed by Mr. Webb and myself on May 7, 1894) have done much good. I have not known him to remain so long free from asthma for years. He has also less catarrh and dyspnœa."

Mrs. S., 33, consulted me for dyspnœa of seven months' duration. She had been always subject to heavy influenza colds, but these had been of quite temporary nature. Four months previous to the onset of the dyspnœa, in July, she was seized with severe attacks of sneezing and a bad cold, which she could not throw off. Then in October began this dyspnœa and cough which kept her to a bedroom for fourteen days, orthopnœa supervening. On recovering, the sneezing was practically gone, but the cough and dyspnœa continued, though she remained fairly well through the winter. At Lady Day she changed house and caught a fresh cold, and her



dyspnœa became incapacitating, and for this she sought my help. I found rhonchi and sibili throughout the lungs, with feeble breath sounds and deficient percussion note and vocal resonance over the upper half of the left lung. Both the second sounds of the heart were accented and its area slightly increased. I gave her iodide of potassium with stramonium and nux vomica. In a week she came again; the dyspnœa had almost gone and both cough and expectoration were much easier, but her sneezing had returned. She was much better in herself. For the sneezing I gave her a 2 per. cent. solution of cocaine, telling her to pour two or three drops in her hand and sniff them up each nostril at the onset of an attack. A fortnight later I saw her again. She appeared quite herself. I found no moist sounds in her lungs. She told me that two or three applications of the cocaine solution had permanently stopped the sneezing. In several other cases I have similarly proved this capacity of cocaine to alleviate irritability of the naso-pharynx, but it must be used with the greatest caution, for its effects upon the nervous system are often most irritating and debilitating, the wonderful and immediate relief it affords making it all the more dangerous a medicine to place in the hand of a patient. On this point I cannot do better than refer you to a "personal narrative" by Professor Windle, which you will find at page 259 of the second volume of the *Birmingham Medical Review* for 1888. The ill effects in his case were so severe that he was forced to give up its use, though it had proved a "perfect palliative" in a most severe attack of hay asthma.

What was the cause of the dyspnœa in Mrs. S.'s case? Her acute attack was probably a broncho-pneumonia which had left its trace in some fibrosis of the left lung, in some chronic catarrh of the tubes and of the nasal mucous membrane. The fibrosis would

diminish the respiratory reserve, whilst the catarrh would render the parts affected unduly sensitive ; thus a slight irritation would be likely to set up unduly great reflex action of the bronchial muscle which the fibrosis would enable the lungs less easily to withstand. Moreover, the heart was weak, so that a slight decrease in the entrance of air into the lungs would be sufficient to hamper the right ventricle and so add to the dyspnœa.

The iodide and stramonium lessened the tubal sensitiveness, whilst the nux vomica strengthened the heart ; but iodide has a tendency to stimulate the secretion from the nasal mucous membrane, hence the return of sneezing.

I would have liked to have related to you still other cases, especially such as would have exemplified the occurrence of dyspnœa from evil metabolism, types which run indistinguishably into that form which is commonly called gouty asthma, and all of which are of surpassing interest ; but this paper is already much too long, and I have intruded unwarrantably upon your time. Forgive me ; and, if I have been wearisome, do not allow such weakness of mine to obscure from your sight the enthralling interest of this great symptom, dyspnœa, which for me has a continually increasing fascination.



## II. THE VARIETIES OF THE EXPECTORATION IN PULMONARY PHTHISIS : THEIR SIGNIFICATION AND TREATMENT

### **Explanatory Notes.**

By "Pulmonary Phthisis," I simply mean any wasting of lung tissue. Thus, under this term, I include, amongst others, the wasting of lung tissue which results from the disuse and collapse brought about by a long-continued serous effusion ; and that wasting of tissue which is the consequence of a prolonged inhalation of metallic or stony particles. I shall only consider those varieties in which I believe the expectoration to have a diagnostic value.

In each I shall state—I. The nature of its expectoration ; II. The reasons for this nature ; III. The treatment of its expectoration ; referring to these heads by the numerals I., II., III., respectively.

### **Chronic Tubercular Phthisis.**

First Stage—**Consolidation.**—I. There may be none. It may be scanty and watery, and perhaps occasionally blood-tinged. It may be in moderate quantity, and

bronchitic in quality, and this too may be blood-tinged. There may be no ordinary expectoration, but occasional slight hæmoptysis. "It is colourless, frothy, or *mucilaginous* in appearance ; if this last there will be a small deposit of a thickish grumous substance with a pale greyish tint not unlike the mud often seen in barley water ; I have never seen this except in phthisis" (Walshe). It will contain leucocytes, epithelial cells, fibrin and *débris* in varying proportion according to circumstances. It may show fine, deep-yellow, sharply-marked stripes very rich in cells. It may, but this is perhaps more especially the case in the third stage, vary with the onset of diarrhœa, and *vice versa*.

II. There is usually none previous to the income of physical signs, the amount of deposit being too small to set up sufficient irritation (see also Pollock) ; there may also be none when the physical signs are well developed on account of the deposit being non-irritative, lying as a quiescent nucleus amidst the healthy tissue, and here cough also is usually absent. Dr. Pollock thinks the expectoration is but of slight prognostic value in the chronic first stage. "The invasion once over, and the deposit tolerated, there may be either—first, simply a lowered vital state seeming to indicate a past illness, and here there would be no expectoration, or perhaps occasional\* and rare, chiefly of mucus ; second, reduced strength and sub-febrile attacks, showing a lung irritated by the deposit ; this deposit probably under-

\* Bacilli are probably always to be found in this occasional sputum.

goes slight periodic increases, and here there would be some expectoration, especially at the periodic attacks."

Niemeyer tells us that cough and expectoration in many cases precede phthisis by a shorter or longer time, and are in such cases due to the prodromal catarrh, and that this catarrh by spreading to alveoli leads to catarrhal pneumonia, and by cheesy metamorphosis and subsequent disintegration to phthisis (see also Jaccoud, p. 150 of English trans.), this catarrh lasting from two or three weeks to years before phthisis is induced.\* If, he says, you find fine deep yellow stripes, very rich in cells, mixed up with the catarrhal expectoration, be on the look-out, as this intimates catarrh of the smallest bronchi; and if, further, blood be intimately mixed up with the slimy or muco-purulent expectoration, then probably the pneumonic process has commenced.

Streaked or tinged sputa occurring in bronchitis are very suspicious: of 25 cases of bronchitis quoted by Walshe 19 had none, and in the other 6 tubercle had been suspected, and was proved to exist *post mortem* in 2. Acute pneumonia with hæmoptysis of pure blood is almost certainly connected with tuberculous disease. Ulceration of the larynx other than tubercular frequently produces streaks. With imperfect irregular

\* I still believe that chronic catarrh, typical or otherwise, as well as pleural and fibrinous inflammation, may be followed not only clinically but *pathologically* by the cultivation of the tubercle bacillus—such cultivation having been impossible prior to that lowering of tissue-life in the affected part which has resulted from the non-tubercular inflammation.

menstruation streaks perhaps, says Dr. Walshe, do not mean much, though they have always occurred where there was a suspicion of tubercle. He says too they are rarely or never the first symptom in tuberculous disease. I believe, however, with Drs. Reginald Thompson and Jaccoud, that extravasated blood may, though very rarely, act as the first starting-point of a chronic pneumonic process leading ultimately to excavation, though whether, as Jaccoud seems to assert, it can produce those special agglomerations of inflammatory cells called tubercles I cannot say.\*

If it contain many leucocytes and much *débris*, but few epithelial cells, then you have probably to deal with an alveolar catarrh (equivalent to a transient cuticular desquamation—Ewart), and may usually expect recovery. If the epithelial cells be many more in amount, then it is probably a catarrhal pneumonia (equivalent to a dermatitis—Ewart) where the vascular layers which grow the epithelium are chiefly affected; here therefore local recovery is impossible, and softening usually results. If in addition there be much fibrin it is a case of caseous pneumonia, and now the resulting expectoration greatly depends upon whether this recent fibrin be re-absorbed (as sometimes occurs, when of course it would not appear), or is involved in the caseous destruction, as usually happens.

As far as I have seen, a hacking cough nearly always precedes expectoration. This cough I believe to be a reflex result, partly due to the delicate respiratory

\* No doubt it can. It is another cause of lowered tissue vitality. *Vide* preceding note.

mechanism being put somewhat out of gear by the presence of the inelastic, incompressible deposit, and partly to the increased sensitiveness of the nerve-endings consequent on the concomitant hyperæmia. A scanty watery expectoration is I think chiefly caused by this cough. It no doubt comes principally if not entirely from the tubes. Now the swift exit of the coughed-up air through these would make them "water" in the same way that one's eyes "water" when exposed to a strong wind. This "watery" discharge is the expectoration.

When the expectoration is less watery, and more mucoid (stickier and stringier), there have probably been previous bronchitic attacks, the patient is peculiarly liable to bronchitic catarrh, and this has now entered upon the stage of alveolar catarrh; or else it is due to a more chronic and serious irritation in the bronchial mucous membrane owing to the exciting cough having been more violent or of long duration.

If blood-tinged sputa come after a prolonged and violent fit of coughing put them down to the mechanical injury produced by this; but if they come "of themselves" then suspect that the tissues immediately around the deposit are irritated by it and consequently hyperæmic—*i.e.*, in the first stage of a chronic pneumonia.\*

\* Longer experience makes me very chary of admitting mere hyperæmia as the cause of even "stained" sputa. Any staining worthy of note is probably always due to tissue degeneration which involves the walls of capillaries or larger vascular twigs.

It is important to remember that two common causes of blood-stained sputa, or a teaspoonful even of blood, are chronic pharyngitis,



Dr. Douglas Powell considers this rusty (tinged) expectoration to depend largely upon constitutional peculiarities, though partly also upon the intensity of the inflammation and the amount of congestion, but that is by no means a constant companion of such inflammatory attacks.

Occasional slight hæmoptysis (pure blood, not mere tinging) points to abnormal disturbance at the periods of its occurrence, such as would arise from slight chills or unusual exertion, &c. It may be merely a more intense condition of the above hyperæmia, but is probably usually due to the giving away of some vascular twig in the diseased tissue from the hyperæmia or a sudden strain put upon it.\* If no other symptoms follow (*e.g.*, increase of expectoration, rise of temperature, &c.), look upon it as a safety-valve and self-curative; but should the expectoration be increased, temperature rise, &c., then the exuded blood has itself acted as a fresh foreign body and has set up mischief around such of it as has not been expelled.

III. Expectoration nil, treatment nil; but if there be cough be careful to sooth it as much as possible, for cough brings expectoration. This dry, hacking cough is best treated by general sedatives, such as opium or

and a relaxed state of the buccal mucous membrane, which is especially common in young girls, and causes them to spit a little lake-coloured fluid on awaking in the morning, but stained sputa of this nature is not aerated, has a mawkish fœtid odour, and is never bright red. One gentleman under my care with chronic throat trouble had spat such streaked sputa off and on for nine years.

\* See previous note.



bromide of lithia, which act both on the nerve-endings and nerve-centres ; if the patient be neurotic, by chloral, which acts chiefly on the centres ; and if he be phlegmatic, by belladonna, which deadens the sensibility of the nerve-endings that have been unduly excited by the hyperæmia of the diseased tissue. These are best given in small doses three or four times a day, the largest to be taken at bedtime. It is equally important to keep up a persistent counter-irritation, as by daily painting the skin covering the diseased portion of lung with lin. or pig. iodi,\* or by keeping a blister open with savin ointment.† Many patients love a linctus,

\* Of the Brompton Pharmacopœia. Its composition is :

Iodi,	gr. 240
Potassii iodidi,	gr. 120
Spiritum rectificatum, ad	fl. ʒij.

This pigment should never be used without previously warning the patient of its possible effect. This effect I have found very difficult to foretell ; the skin, which appeared thin, of some fragile, wasted woman, I have seen resist its repeated application, whilst that of a burly navvy has given way somewhat deeply on the first. Complexions cannot be relied on, the blonde may obstinately withstand, the dirty sallow yield at once. From a therapeutic point of view, however, there is no need for apprehension ; one application never does too much, very seldom enough. An open sore kept slightly discharging for two or three weeks I have several times observed to be most beneficial, greatly lessening the cough and expectoration. A skilful nurse will keep the sight of it from her nervous patient till the day when it is to be allowed to heal, then show it him from time to time. It is quite touching to see how its gradual vanishing fills the patient with eager hope that his internal lesion must be keeping pace with its external relation.

† Iodised oil (ol. olivæ 93, iodi 3) is a very pleasant substitute for the linimentum iodi on delicate skins. It barely stains skin or linen.

and are more satisfied if sedatives are given in this form. One of the best I have tried is—

R Liq. morph. acet.,	℥8.
Æther. chlor.,	℥3.
Succi limonis,	℥15.
Mucil. acaciæ, ad	℥60.

It may be given three or four times a day, but especially at bedtime ; in fact, all sedative drugs are chiefly needed at this hour, change of position and atmosphere intensifying the irritability. But it is most important at this stage to prevent any undue exercise of the lung which would make taut the walls of the air-sacs and fill them fuller with blood, thus intensifying their perception of the deposit ;\* also to avoid exposure, which would prove injurious to the tissue immediately surrounding. For it must not be forgotten that those alveoli (*e.g.*) which are next to the deposit have one wall practically formed by this, that is to say, one wall is immovable ; they cannot, therefore, collapse and expand as they should, are unable, in fact, to take proper exercise, and live considerably below par on insufficiently aerated blood, and consequently are peculiarly susceptible to evil external influences. The thing to do here is to tide over the delicate time whilst the lung is becoming accustomed to what are to be in future its new conditions of existence ; this done, these

\* This is true ; but on the other hand, without lung exercise we cannot hope for such tissue vitality as will produce “curative” resistance. By “undue” is meant sudden or violent exertions for the individual in question.

wasted and paralysed cells may become as invulnerable as their better situated but more sensitive fellows, for one not infrequently sees lungs, recent disease of which has killed their possessor without relighting the quiescent focus of an old mischief.\*

The catarrh, whether this exist previously to any deposit, or as one of its consequences, is best treated as a primary attack of mild subacute bronchitis.† If there be scanty and very watery expectoration, the cough is probably its sole cause, and special attention should therefore be paid to it. If the sputa be tinged or streaked with blood any congestion of the liver or pelvic organs should be removed, and the bowels kept freely open by mild laxatives, such as Hunyadi Janos or Friedrichshall water; it is here too that counter-

\* In our gloomy prognostications as to the lack of durability in the condition called arrested phthisis we do not pay sufficient heed to this undoubted fact. We know that most acute outbursts in these cases are due to rupture of a quiescent nodule. Hence, we regard *all* such nodules as gifted with a dread potentiality which is bound sooner or later to become kinetic. But this is not so. Probably, for each one which is so fatally transformed, at least 19 remain quiescent, and finally, losing their potentiality, become extinct.

† The following will be found a most valuable prescription for this condition :—

R. Extr. glycyrrh.,	
Pulv. sacch. alb., āā,	gr. 180.
Pulv. acaciæ,	gr. 360.
Pulv. tragac. co.,	gr. 45.
Olei anisi,	℥ 30.
Aq. rosæ,	q.s.
Misce : fiant trochisci sexaginta.	

irritation is especially indicated. After any hæmoptysis rest on a couch should be insisted upon during the next twenty-four hours, and the above treatment carried out ; the temperature should be taken every four hours, and if at any time it reach  $100^{\circ}$  F., the patient must go to bed and stay there till it become normal again. He should be encouraged to cough if the after-sputa be blood-stained ; his danger lies not in the loss of blood, but in the irritation produced by the blood remaining in the hypersensitive air-cells.\* Persistent efforts must be made to reduce the temperature ; cough and expectoration nearly always vary directly with this ; if the elevation be slight, Niemeyer's pill† should, I think, be given the first trial ; but if the elevation be greater, and moreover not hectic but continued, then antipyrin will usually prove the best drug—gr. 10 to gr. 15, three or four times a day.

Second Stage—**Caseation**.—I. "In chronic cheesy degeneration before excavation has begun, the sputum is almost entirely of catarrhal secretion from the bronchial membrane, and hence, at any rate to the unaided eye, is the same as that of an ordinary catarrh. It may contain though a few elastic fibres. If blood be mixed in reddish specks or streaks with the expectoration for long together, or frequently recurring, we may infer

\* And of the passage of the blood (with infective matter) into distant parts of both lungs.

† R Quin. sulph., gr. j  
 Digitalis, gr.  $\frac{1}{2}$   
 Extr. opii, gr.  $\frac{1}{4}$

Fiat pilula : to be taken three or four times a day.

with almost absolute certainty the existence of chronic caseous infiltration, even though there be no distinct physical signs." (Guttmann.)

As a rule, the inroad of caseation, if it be at all general and unaccompanied by increasing consolidation or commencing excavation, is heralded by a lessening or disappearance of the hacking cough; the watery, scanty expectoration becomes thicker, more mucopurulent, easier of expulsion, and less in quantity. It is an oasis between the wearing irritation of consolidation and the destructive advent of excavation; but it is an oasis seldom seen in the adult, for excavation and consolidation usually overlap one another.

II. The expectoration is easier because the softening masses offer less and less resistance to the compressing expulsive efforts, the air-cells affected have become resigned to their fate, and no fresh ones are being involved. It is thicker, &c., because the cough being less, the tubes are less irritated, and because their lining is more or less diseased, made less sensitive, from their previous exertions. The acute bronchitis has gone; they are now in the chronic stage.

III. Little needs to be done as far as regards expectoration; the treatment consists chiefly in warding off excavation; the longer this second stage exists the milder will be the inroad of the third.\*

Third Stage—**Excavation.**—1. Opaque specks and purulent streaks appear in the expectoration, "their opaque, pale-buff or yellow tint, contrasting with the

\* Because encapsulation will be gradually taking place.



lighter coloured and more transparent mucous body of the sputum, which grows less and less aerated." (Walshe.) As it becomes more purulent it has a tendency to be nummulated. Now these nummuli are merely agglutinations of expectoration varying from loosely cohering, slimy, flat, discoid gobbets with smooth superficies to "small pellets with jagged, sharply cut outlines, opaque, semi-floating, non-aerated, and of a dirty," whitish-yellow "colour (boiled rice sputum)." (Walshe.) It is usually more easy of expulsion and larger in amount than during the first stage.

From the earliest period you get elastic tissue with sometimes fragments of minute bronchi or vessels. According to Dr. Douglas Powell, elastic tissue is to the expectoration what humid crepitation is to the stethoscope. Expectoration is usually especially copious in the evening on lying down and on waking and rising in the morning. It often disturbs patients at night from its rapid accumulation, rest and sleep coming back again so soon as it is spat up. It may become fully purulent at once, instead of a period of specks and streaks intervening; it may be very difficult of expulsion, and even scanty; sometimes with comparatively scanty crepitation there is abundant and easy expectoration, or the opposite—a case of much cry and little wool, and *vice versa*.

The softened mass may clear out in almost a single night with the sudden expectoration of much purulent matter, or the vomica may take months to form; the



rapidity of excavation is by no means proportionate to the amount of expectoration ; a large cavern may clear out and dry with a history of expectoration which is only to be obtained by the most careful questioning, and again a pint a day may be spat up for weeks, yet no crepitation sufficiently large to be termed cavernous produced ; in fact, as Guttman tells us, "generally in phthisis the quantity of sputum depends on the intensity and extent of the accompanying bronchial catarrh."

Frothiness of expectoration by no means necessarily indicates difficulty of expulsion. In some cases I have seen where the sputum was completely hidden by a thick layer of froth (like the "head" on well-poured-out draught beer) the act of expectoration was very easy, and the amount very large. In one case I remember it was nearly entirely brought up during two or three periods of the twenty-four hours.

"'Tis astonishing that septic decomposition is almost always absent." Cholesterin crystals may occasionally occur. The consistence of the expectoration varies greatly ; it may have the fluidity of pure pus, the dense nature of clotted blood, or re-acquire the tenacity and wateriness of the first stage from an intercurrent bronchial catarrh.

This is the stage of hæmoptysis *par excellence* ; if during consolidation it be regarded as dangerous, then is it more dangerous during caseation, and most so in the period of excavation ; in amount it varies from periodic streaking or tinging to mortal and sudden.

There is a rare form of expectoration mentioned by Dr. Pollock, of which I have only met one case ; this is the cretaceous, bony, or horny variety.\* It is, he says, an indication of chronicity with attempt at cure. You may meet with it in people of eighty, it being then due to tubercle formed by fault in sanguification at puberty,† and which has since remained dormant without any symptoms. He describes two modes of its occurrence ; first, when after an attack of ill-health, a few cretified masses are spat up with no further symptoms (as in the case I saw), this no doubt happening to numbers of people ; secondly, the more frequent, when there are successive inroads of tubercle with concomitant chalky expectoration occurring fitfully, as well as the continuous ordinary purulent variety, showing that the disease is advancing slowly with repeated attempts at cure.

II. The discharge is purulent, just as other discharges are when these are the result of tissue waste ; the more rapid the excavation the more purulent will

\* If we look at the number of acts of expectoration, then those which result in chalky sputum are extremely rare. But the proportion of phthisics who expectorate cretaceous sputum at some period of their life is by no means small. It is rare for the same individual to bring up chalky matter more than two or three times in his life, and even these are unlikely to occur whilst under phthisical treatment : for chalk = arrested or extinct tubercle. I have several times noted it in people who were patients for other causes.

† Not necessarily at puberty, but at any previous period of life. Indeed it is more likely to have been formed during adult life, for tubercle occurring during adolescence is less likely to heal than that arising later. Not necessarily from "fault in sanguification" (anæmic debility), but from any cause which renders the lung tissue fertile soil to the bacillus.

it be, unless indeed the work of destruction progress *too* rapidly, when fragments of tissue may be undermined and detached which have not had time to become converted into pus. (*Vide* Acute Phthisis, p. 80.)

The amount of mucus indicates the amount of tubular irritation and inflammation; this may be due to, first, the disease having originated as a broncho-pneumonia; secondly, the constant cough of the first stage; thirdly, the irritation caused by the passage of pus through the tubes. If it be produced by the first or second of these it is no new product but merely a continuance of what we have noted in the first and second stages; if by the third, then at first we get an almost pure, purulent discharge of small quantity, and with merely sufficient cough to expel it, but gradually mucus begins to appear, whilst the cough and expectoration increase from bronchitis set up in the healthy tubes by the irritating discharge. But after a while the amount of mucus lessens, for the tubes either get chronically degenerated, and thus themselves excrete a more or less purulent fluid, or else they become accustomed to the passage along them of the morbid material from the cavities and cease to be sensibly irritated by it. In addition to this the cavities as they clear out have their walls partially formed by sections of tubes which at first continue to pour out their mucoid secretion, but eventually break down into pus. Hence it happens that, as excavation goes on, the expectoration gets less and less aerated.

Nummulation is hard to explain. A fluid sputum

lying some time in a cavity after it has been secreted, naturally loses some of its fluidity, this being partly dried off by the circulating air, and partly, no doubt, re-absorbed ; thus, what remains is solider and likelier to cohere and retain its form, and is therefore spat up in a lump. That this is the explanation of a great deal of nummulation seems to me the more probable for these reasons : first, in the rapid disintegration of acute phthisis you rarely, if ever, see true nummuli ; secondly, when a cavity is drying up—*i.e.* its secretion lessening—the expectoration gets more and more nummulated ; thirdly, if there be much admixture of mucus there are no nummuli ; because, no doubt, the tubular sections exposed in the wall of the cavity keep its contents moist and liquid by their, as yet, mucoid secretion. On the other hand, the most typical nummuli are lymph masses floating in a watery sea ; evidently here lymph has been secreted, not pus ; hence it is an evidence of very slow *secretion*, but the mass once secreted may be, or may be not, at once spat up. One would expect this to indicate an old drying-up cavity and the watery sea strengthens this supposition, for Lebert says the older the cavity the waterier its contents. These are the “boiled-rice” sputa. It is this state of the sputum which is especially prone to form casts from the toughness of its fibrin. Again, Dr. C. J. B. Williams tells us that albumen itself clots as secreted, forming opaque flakes ; hence here is evidently another cause of nummulation. He also says that when this curdy clotting of albumen occurs to a

great extent you may get casts, though no doubt these chiefly result from fibrinous secretion. I have several times noticed tiny pellets of this variety in chronic slight cases where the patients appeared in fair health.\*

The expectoration is easier, because as the hard resisting deposit vanishes the lung yields more readily to compression, especially since, in place of consolidation, you get air-filled cavities which collapse on the slightest pressure ; or rather, speaking more strictly, are only kept distended by the thoracic walls and their own. It is more in amount because we have added to the previous secretion the caseous contents of the clearing-out cavity, increased secretion from the bronchi irritated by these contents, and later, secretion from the cavity's walls.

Elastic fibres are the last elements of the lung tissue to be destroyed ; therefore it is that they generally resist destruction in the lung, and are spat up unchanged. I have, however, failed to find them in two or three cases of undoubted excavation. Their presence proves the existence of excavation ; their absence does not disprove it.† According to Dr. Ewart, the moister the process the more elastic tissue is there to be found in the expectoration ; but even true dry caseation does not completely destroy these fibres, though it tends to make them brittle, and so comminute them. Hence their absence may negative excavation in cases of catarrhal pneumonia, where it is characterised by

\* Nummulation is not confined to phthises : it occurs in chronic bronchitis and acute pneumonias.

† Cf. the similar value of tubercle bacilli.



inveterate œdema with maceration of the deposit. He further says that in fully formed cavities we do not habitually find any elastic sediments amongst their contents. Elastic fibres may come from the finest bronchi or from the alveoli. Dr. Guttmann says the formerly held opinion that the long straight or slightly coiled ones come from the bronchi, and the reticulated ones from the alveoli, is now generally admitted to be erroneous. (But *vide* Bronchiectasis.)

The expectoration is copious on going to bed, partly from the energetic breathing induced by this act, partly from the change of position ; the former quickens the lung's life, and so intensifies its perception of the sputum ; it also tends to produce partial vacua, which, with the friction caused by the rapidity of the air-current, excite movement in the sputum. The latter brings gravity into play, causing the sputum to roll over fresh surfaces, these surfaces being unaccustomed to it, communicate its presence to the sensorium, and it is then coughed up. This sensitiveness of fresh surfaces is well shown by the fact that many patients do not cough and expectorate if they acquire the horizontal position very gradually, thus allowing the sputum to invade these surfaces gently, bit by bit ; an action often adopted by the patients themselves, and one which I have frequently recommended with success to some whose secretion was slight, but lung sensitiveness extreme. Similarly with rising in the morning ; but here we find another cause in the long stillness of sleep which has allowed the steadily secreted



sputum to increase unconsciously and unirritatingly throughout the night.

Patients who secrete large quantities of sputum are often disturbed once or twice in the night for the purely mechanical reason that the lung cavities overflow; they wake, expectorate a quarter of a pint or more of sputum with very little trouble, and at once have ease and go to sleep again. On the other hand, there are many trying cases with scanty expectoration who ceaselessly cough for two hours or more before the new surfaces will allow any secretion to lie upon them, also other still more trying ones where no part of the lung will tolerate any secretion, and hence we have coughing throughout the twenty-four hours, and the sleep broken every hour or every half-hour with fits of hacking. In these cases of hyperæsthesia I believe there is always either much surrounding hyperæmia or else an inflamed cavity.

Scanty expectoration of pus generally implies difficulty; it shows that the hard deposit is being but slowly removed, and hence the mechanical conditions of the first stage but slowly changed; the secretion being slow the expectoration is dry and hence difficult; its slowness also gives time for contraction and fibrosis to take place in the peribronchial sheath of the cavity, making its walls unyielding. But difficulty of expectoration also depends largely upon the size of the bronchial orifice leading to the cavity, upon the relative position of this orifice—whether it be at the bottom or top of the cavity, upon the direction of the bronchus

(the cavity's drainage tube)—whether it be at right angles to, or merely a continuation of the cavity, and lastly, upon the position of the cavity itself with regard to the whole lung. This last cause plays an important part in the final stages of the disease where the expulsive efforts are too weak to overcome the action of gravity.

The amount of crepitation audible to the stethoscope depends greatly upon the position of the cavity; if it be central, then all the softer ones are lost, if superficial, every sound is heard with marked distinctness. But apart from this, some lungs are very tolerant of their secretion, and, even though its formation be slow, they allow it to fill the cavities, merely expectorating the overflow. In these cases the amount of expectoration puts us on our guard against concluding that excavation varies directly as crepitation.

An area of consolidation usually first softens where it is exposed to the air—*i.e.*, at the termination of some bronchus—as is proved by the many half-excavated cavities found in the post-mortem room; here, too, then, the excavation would begin, and the *débris* be spat up piece by piece as formed. But a very common method of excavation (Ewart) is that in which the softening occurs *last* at the bronchial termination; hence, when this gives way the whole area is at once cleared out. If the cavity be a large one, and full of fluid pus, it may resemble an empyema opening through the lung (for diagnosis, see *postea*). This is a favourable, because a rapid method. Rindfleisch thus explains it: during inspiration there is a general traction upon the branches

of the bronchial tree ; as soon as softening has sufficiently advanced, a separation is thus effected between the root of the softened mass and the bronchus which passes into it ; air is then at once drawn into the vent thus made, and the contents escape.

If the cavity clears out without setting up any irritation then the expectoration need be but very little, for very few cavities can contain a pint. Should the expectoration of this pint (or less) be distributed over some months, we can easily understand how it might fail to attract the patient's notice. I remember a professional tenor who must have been taking the part of Faust, daily, in the Manchester Free Trade Hall during the most active period in the excavation of a cavity in his right lung, which reached from the apex to the 4th rib and inferior angle of the scapula respectively.

But unfortunately excavation usually sets up much irritation, both around the cavity and in the bronchial tract over which the pus passes, and hence the large amount of expectoration. We have a somewhat analogous action in the immense psoas abscess which may result from the partial destruction of one vertebra.

When the expectoration is very frothy, and at the same time easy, I think the source of at any rate a considerable portion of it must be very distant from the large bronchi, that to reach these it has to pass through many air-traversed tracts, and that these tracts are not very sensitive to its presence. I remember a man who showed me every morning about half a pint of expectoration, which he had coughed up with comparatively

little trouble in the one to two hours since rising from bed. It always had a head of froth a good half-inch thick upon it, and I never saw any of this man's expectoration at any time in the day which was not similarly hidden. Both his lungs were full of large loose crepitations, but there were no true cavernous sounds. They were probably riddled with small excavations. Here the free purulent discharge overflowed from the most distal excavations into tracts through which air passed to other terminal portions of the lungs, and was allowed to lie in these tracts till it was thoroughly aerated.

The freer the air supply the more marked is the absence of septic decomposition. Is it because there is too great an abundance of oxygen to allow the micro-organisms to grow? Still, though the more difficult the expectoration the more liable are the secretions to become offensive because less air enters and the products tend to lie stagnant, still even this stagnation cannot spontaneously originate septic decomposition; its source appears always to be outside the lungs. (Ewart.) On the other hand the lungs are pre-eminently self-infective. Distal segments of tubes which traverse cavities often display in a most striking manner groups of consolidation, caseation and excavation arranged in little circles round their terminal bronchioles, spreading arborescently, so as to allow no doubt as to their mode of origin.

Occasionally one comes across cases where there is a copious purulent expectoration for months together with no change, or scarcely any, in the physical signs.

The bronchioles, alveoli, and excavated portions of lung have taken on a debased pyogenic energy. It is in many ways analogous to the condition of the trachea and large bronchi in cases of "Winter Cough."

What I have already stated sufficiently explains the variation in the consistence of the expectoration; it simply means a variation in the activity—temperament—of the disease.

Hæmoptysis requires a thesis to itself. Streaks indicate congestion as in the first stage. Excavation often denudes arterioles of their supporting tissues, or destroys the wall of one of them. The chief danger is from the bulging of an arteriole's wall into a cavity, the consequent formation of an aneurism, with finally, the bursting of this aneurism.

III. The chief indication here is naturally to endeavour to stop the progress of excavation, but the methods employed for this purpose belong more to the general treatment than to that of expectoration. As a rule they are unsuccessful\* until the caseous mass has been completely removed. Still it must never be forgotten that very few of these caseous masses weigh even two ounces, and that very few cleared-out cavities can hold half a pint. It is therefore the expectoration of irritation which forms nine-tenths of the bulk of the sputum, and none of this is absolutely necessary, for we

\* Fortunately for the patient. My former self is here in error. The caseous masses are only sources of evil in the lung and the sooner they are got rid of the better. But we must limit the progress of excavation in this sense, viz., prevent the *débris* setting up mischief elsewhere in the lungs during its expectoration.



know that in fortunate cases it is quite absent. It is simply a drain on the system producing amyloid\* troubles and debility and should therefore be stopped. It chiefly comes from the tubes which are usually in the same condition as they are in chronic bronchitis and should be similarly treated; such a mixture as this I have found most generally useful:—

R Tincturæ scillæ,	℥ <sub>20</sub>
Tr. camph. co.,	℥ <sub>30</sub>
Sp. æther. nit.,	℥ <sub>30</sub>
Liq. ammon. acet.,	℥ <sub>120</sub>
Aq. camph., ad.,	℥ <sub>j</sub> . t. d. s.

Continued counter-irritation should also be kept up over the seat of the lesion to allay the congestion beneath. Jaccoud strongly advocates the internal administration of creasote in conjunction with some preparation of salicylic acid. Creasote and its congeners are certainly the drugs from which I have seen the best results; and seeing the great and rapid benefit derived by inhalation of salicylate of zinc in relaxed sorethroat† one would expect good results from it in chronic tubal irritation. But Jaccoud also insists on the inhalation by means of the steam spray of carbolic acid; the patient is to begin by inhaling 4 oz. of a 1 per cent.

\* This is one of those legendary dicta which are handed down by generation after generation of text-books. I did not believe it in 1883: I don't in 1894. The large majority of the degenerated livers of phthisis are fatty and not amyloid. (*Vide* "La Phthisie Pulmonaire," by Hérard, Cornil and Hanot, 2nd ed. p. 255.)

† R Zinci salicylatis, gr. 20

Aquæ, ℥<sub>j</sub>

To be used in a steam spray with two ounces of water twice a day.

solution daily in three or four sittings, and this is to be gradually increased till 20 ounces of a 2 per cent. solution are daily inhaled. This proceeding, he says, almost always takes away fœtor and lessens expectoration, and sometimes dries a secreting cavity. My experience unfortunately has been very different, very seldom have I seen the desired effect attained. For instance, as to fœtor, in three cases of empyema opening through the lung, I used it with no effect; in none did the patient's neighbours think the fœtor lessened. I have purposely smelt the breath of patients whilst they were using the inhalation, and have at the same time examined their sputum, but never have I found either to be much less offensive. The inhaled vapour seems incapable of attacking the fœtid masses, it simply plays around their exhalations, partly destroying the fœtidity of these, whilst the inside of the masses it never touches; consequently when these are expectorated, they are as offensive as ever. So, too, if we are to prevent the reproduction of developed bacteria or their spores, the air must be kept constantly saturated with a solution of carbolic acid, which is stronger than 1 in 42.\* Is it possible to do this in the case of the minute bronchi and diseased air-cells? If not, then it is hopeless to try and disinfect a lung by the inhalation of carbolic acid. It seems to me very doubtful that any inhalation can enter much beyond the two main bronchi. Personally, I have never been able to feel the warm glow of the atomised fluid for any considerable

\* Brunton's "Pharmacology," p. 73.

distance beyond the bifurcation of the trachea ; I can just say that the roots of the lungs told me of its presence there. I should say then, if the expectoration is lessened by it, this only refers to that coming from the large tubes, or else the lessening is due to the absorption of the acid into the system. But supposing the acid does reach to the uttermost parts of the lung, it can only do so in a state of extreme dilution, no more concentrated in fact than it could be taken up by the blood from the stomach if it were administered internally. Other things being equal, a drug is much more likely to act on the excreting surface of an ulcer when supplied by the blood than when simply wafted over this surface by the surrounding atmosphere, and I have certainly seen better results from tar pills than from the inhalation of carbolic acid.\* Sedative inhalations stand in a different category ; it is the larynx, trachea and throat which are often the chief disturbers of sleep and producers of harassing cough, and these no doubt can be well reached by inhalations. It matters little, however, what these contain ; unmedicated steam I have usually found as beneficial as most, except perhaps the compound chloroform vapour† of the Brompton Pharmacopœia.

Inhalations have been advocated in order to prevent the patient from infecting his healthy neighbours ; the

\* Vide Essay on "Antisepsis in Phthisis."

† R Chloroformi,	℥ 10
Succi conii,	℥ 60
Glyc. ac. carb.,	℥ 50
Aq. bullientis,	℥ viij. Inhaletur vapor.

tabulated experience of the Brompton Hospital\* is, I think, quite sufficient to show the needlessness of this except in the case of such close companionship as that of husband and wife, and to these I should say—put not your trust in vapours.

The spittoon should always contain disinfecting fluid, and it should be emptied three or four times a day.

Objections to inhalations are these:—the impossibility of getting the patient to use them for more than one or two hours in the twenty-four; the increased exercise of the tissues which they necessitate (Douglas Powell); and, in the case of medicated respirators, their unsociability and unsightliness.†

Should the cough be very harassing and the expectoration very slight, with but scanty crepitation, then soothing lincti are admissible; so too, though excava-tion be even rapid, if the patient be suffering much from want of sleep on account of cough and expectora-tion, an evening sedative must be given. Perhaps the best is a sixth of a grain of morphia with a hundredth of a grain of atropine, given hypodermically; this is less likely to produce morning headache than morphine alone. Should headache come a dose of spirits of ether and ammonia will usually remove it. But whilst

\* Dr. C. Theodore Williams in the *Brit. Med. Jour.*, Sept. 30, 1882.

† For they must cover the nose as well as the mouth: otherwise the patient will breathe through the unguarded nose. Mayer and Meltzer's cellulite oro-nasal respirator is the best I know of.

excavation is proceeding it is a golden rule never to give a sedative during the day, and especially not in the early morning. It is most important that the morning cough should remove the sputum which has collected during the night. Sometimes, however, this is very trying to the patients, utterly wearing them out, and preventing any attempt at breakfast. There may be a prolonged fit of coughing for half an hour or more before any sputum is expectorated, and its removal is throughout very difficult; for this condition fortunately we have an almost never-failing remedy—a drachm of aromatic spirit of ammonia in seven drachms of infusion of senega, given the first thing on waking, enables the patient to remove his sputum with ease and rapidity.

If, with no signs of asthma, the patient complain, and usually at night, of great tightness across the chest, with stifling sensations and inability to remove his phlegm, then a good rub with turpentine liniment, or a mustard and linseed poultice, along with a stimulating expectorant, is usually sufficient to give relief and induce sleep.

A patient cannot be too strongly impressed with the mischief which is likely to result from swallowing his sputum; some form of dyspepsia is certain to result from it, and dyspepsia is the most troublesome of all the ailments incidental to phthisis; there is the chance too of the intestine becoming infected by its means.

If the sputa be streaked keep the patient in bed, keep up counter-irritation, and put him on a course of



rhatany or iron. If the hæmoptysis be severe, Jaccoud goes much farther than this; he applies large blisters with forty to sixty dry cups twice a day to the legs and base of chest, and gives a third of a grain of opium every hour till sleepiness occurs, and afterwards sufficiently often to maintain this disposition. I believe the most important thing is to clear out the bowels and unblock the liver; a concentrated saline purge is the best, this extracts much fluid from the blood, thus lowering the blood pressure and increasing the tendency to coagulation. To stop the actual hæmorrhage I have found nothing of much value; I generally use ergotin subcutaneously, gr. 20 of Bonjean's preparation injected into the buttock, but have seldom been able to trace any benefit to its use. However, I remember when making this remark to the resident physician at Ventnor in 1882, he expressed great surprise, and stated that he always injected ergotin with the greatest confidence in the result. At the suggestion of Dr. Line of the Borough Hospital, who had found it more valuable in hæmorrhagic small-pox than ergotin, I have several times given Ruspini's styptic in drachm doses every hour. I am inclined to think highly of it. In the six or seven suddenly fatal hæmorrhages which I have seen, death was generally due to asphyxia from blood in the tubes and not from loss of blood; so that in the third as in the first stage the patient should always be encouraged to cough the blood up. In fact, so pained was I with these asphyxic deaths that I determined to invert the next case at which I might be present; fortunately,

perhaps, from the patient's point of view, no such chance presented itself during my further stay at Brompton.\*

Towards the death-day, when there is great exhaustion, the sputum often clings about the throat, trachea and main bronchi, causing great distress and sometimes an agonising dread of being choked—the patient plunging her fingers (it is usually a woman) down her throat so as to withdraw the long strings of muco-purulent matter. Happily this wearing distress is capable of great alleviation; stimulants† should be given to increase the coughing power; almost as important is it to clear the fauces with lemon and glycerine, or Wyeth's chlorate of potash discs; but better than these is a linseed poultice with a suspicion of mustard, not more than one line in thickness and some 6 in. by 3 in. superficies, placed over the trachea and renewed at first every ten, fifteen or twenty minutes. This I have hardly ever found fail in giving relief, and it often brings quiet and sleep when the patient is far too weak to bear the weight of the ordinary chest poultice.

Since writing the preceding I have in a number of cases employed the tincture of belladonna in order to

\* Refer to the Essay on "Hæmoptysis."

† R. Sp. ætheris, sp. ammon. arom. āā ℥30; tr. aurantii ℥10; aq. camph. ad 3j, given every 6, 4, or 2 hours does more than any medicine I know toward maintaining life and producing euthanasia. In bad cases I usually give in addition, at the alternate hours, half-ounce doses of brandy mixture, but the great power of ammonia in making the expectoration more fluid, "loosening the phlegm," gives it easy pre-eminence, nor does it appear to lose its power when continued for weeks if one is careful to vary the amount according to circumstances.

arrest secretion where this was in large amount and evidently coming chiefly from chronically inflamed tubes. Frequently this, unaided by any other drug, has proved of marked benefit, lessening the secretion considerably and speedily. Especially have I observed this action in children suffering from pneumonic phthisis or the later stage of broncho-pneumonia ; in them ten to fifteen minims every four hours has, in a few days, almost entirely removed numerous moist râles and, where the child has been old enough to expectorate, reduced the amount of the sputum to a minimum. I have not, however, generally found it efficacious in relieving any accompanying spasm. These remarks on the action of belladonna are in no way new but only a confirmation of many which have previously been made.

#### Fourth Stage—**Cavity formed.**

(a) Quiescent.—I. If thoroughly so then is the expectoration *nil*. Often you have a slight “clearing the throat” of a little muco-purulent matter when the patient gets up in the morning.

II. It is a case of arrested phthisis, and is, happily, by no means uncommon. The inner lining of the cavity wall has taken upon itself to act as any other healthy lining membrane. But, as is the case with all pathological repairs, it never acts so *well* ; it is easily excited into taking on a diseased activity ; and the morning throat-clearing tells you of the cloven hoof.\*

\* And, moreover, in it bacilli can usually be found.

III. Treatment is entirely prophylactic.

(*b*) Extending.—I. The expectoration is that of the third stage.

II. An already formed cavity has broken down afresh : if previously it had been thoroughly quiescent then there would simply be the expectoration caused by this new destruction ; but if it had been one of the varieties mentioned below, then the expectoration of that portion of the lining still intact would be added to the expectoration of excavation ; the two portions would, however, be indistinguishably mixed.

III. The treatment is that of the third stage.

(*c*) Irritated.—I. The expectoration is stained, often frothy, not usually considerable, in consistence watery, expulsion difficult ; with it is generally pain and a trying cough. This is very often more or less modified by the co-existence of the third stage, but I have come across two or three uncomplicated cases where the mischief has supervened in a quiescent cavity, and passed off without further damage.

II. Dr. Ewart divides the wall of a cavity into three layers ; the inner or false membrane, the fibro-vascular layer or inner wall of the capsule of the cavity, and lastly the purely fibrous layer or outer wall of the capsule. Further, the relative thickness of these varies with age and degree of irritation, the second being the real gauge of activity ; if the cavity be irritated, then this layer becomes extremely congested. The cavity has caught cold, its lining is “sore,” as we speak of a “sore” throat ; and, like the throat, this soreness may

go on to ulceration or even sloughing, may be merely transitory, or may continue chronically inflamed, in which case the expectoration would become mucopurulent and lose all trace of blood.

III. Persistent counter-irritation is here especially valuable, *pig. iodi*\* or a blister kept open with *ung. sabinæ* is perhaps the best form, though Jaccoud advocates the use of Vienna paste.† The cough is always in excess and should be restrained. In these cases I have seen Dr. C. T. Williams give the following mixture with great success.

R Ac. phosph. dil.,	℥ 15
Vin. antim.,	℥ 5
Tinct. quassiæ,	℥ 30
Syr. aurant.,	℥ 20
Aq., ad	℥ j.

(*d*) Ulcerous.—I. Expectoration is copious, blood-stained and purulent, mixed with a ropy secretion from the intensely vascular bronchi (Douglas Powell). Dark changed clots may be spat up, or occasionally free hæmorrhage may occur. In a more chronic variety, as in one of my cases at Brompton, it is uniformly stained a dark red, and consists of slimy nummuli bound together with much mucus, there also being occasionally added a matrix of dark bloody fluid. The cough, too, is usually very trying, and militates strongly against the rest which the patient so greatly needs. As the case

\* For composition see note p. 45.

† R Caustic potash, 5 parts  
Quicklime, 6 „  
Water, q.s.



improves the expectoration gets scanty and viscid, till at last it consists of bronchial mucus only, the cavity finally becoming quiescent. But improvement is always extremely slow.

II. It is “*c*” gone to the bad, principally no doubt owing to a debilitated constitution; the sore throat has become ulcerated. But Dr. Douglas Powell scarcely looks upon it in this light, he considers it to be an erysipelatous inflammation. Its depressing effect upon the constitution greatly strengthens this view; it also comes on without any appreciable period of irritable cavity preceding. On the other hand some of these cases get into a very chronic state with no symptoms pointing to an erysipelatous nature, while others have slowly supervened upon an irritated cavity. In fact, just as in faucial inflammation we have all gradations from mere congestion to a sloughing hospital sore throat, so do we find lung cavities exhibiting all varieties from the mildest irritation to erysipelatous ulceration.

III. The general health is always very much impaired; thoroughly good food with pure air, and plenty of both, are therefore amongst the essentials of treatment. A quinine and iron mixture is the best tonic. There is much depressive irritability and systemic hyperæsthesia; opium is *par excellence* the remedy for these. Rest in bed is desirable in “*c*”; here it is absolutely necessary. The cough is far more than is needful and very wearing; special attention should be given to lessening it; if the opium be not sufficient,

soothing inhalations may be of service ; if the patient be too weak to use these efficiently, medicated sprays should be employed ; active counter-irritation, as a rule, cannot be borne.

(e) Gangrenous.—*Vide* Gangrene of Lung.

(f) Secreting or Pyogenic.—I. Constant easy expectoration of pure pus, which comes up at any time of the day, but especially after exertion. No pain nor distress, and but little cough. The pus tends to get more and more diffuent and creamy. The duration is indefinite. Hæmoptysis is extremely rare. The quantity is moderate or scanty, but never excessive.

II. The wall of the cavity has become a pyogenic membrane, as do the walls of other chronic abscess cavities ; and here, as with those, the danger is the same—that of fatty or amyloid degeneration. The thickness of the wall slowly increases ; I have seen one which was a sixteenth of an inch thick, and no doubt some are much more. The secretion goes on regularly and equably, there is seldom any “ bagging of pus.” The size of the cavity is never very large—can never reach anything near the size of a psoas abscess cavity for example—hence, therefore, the moderate amount of discharge.

III. The sole aim here is to stop the secretion of pus. Jaccoud recommends the inhalation of carbolic acid by means of the steam spray ; he begins with 4 ozs. of a one-per-cent. solution, to be taken daily in three or four sittings, this amount to be gradually increased up to 40 ozs. if necessary. He states that lessening of the expectoration is almost constant, and

that sometimes the cavity completely dries. Counter-irritation, he says, should never be omitted. Alcohol and creasote taken internally have, he believes, the same effect as the carbolic spray. At Brompton, I remember seeing a very happy and speedy result from the use of tar pills (gr. 2 t. d.) alone. Ringer and Murrell have shown the efficacy of tar in removing the moisture of chronic bronchitis. Belladonna —  $\mathfrak{m}$  15 of the tincture four times a day—has often proved in my hands a most effectual lung drier. Murrell\* speaks of the result of treating eighteen cases of phthisis with pure terebene, obtained by the action of sulphuric acid on oil of turpentine, as “certainly most encouraging.” His dose is five drops on sugar every four hours, this to be gradually increased to twenty.† These are the cases where intra-pulmonic stimulating injections are especially indicated. Mosler of Greisswald, Pepper of Philadelphia, Shingleton Smith of Bristol, and many others, have tried injecting weak solutions of Condyl’s fluid, bichloride of mercury, iodine and iodoform, but with no definite results. Their chief danger lies in their passing out of the cavity and setting up pneumonia in other parts of the lung; we cannot tell where the bronchial opening into the cavity may be: for aught we know it may be in the floor, so that any injection would at once pass through it. Aware of this, injectors have usually employed such solutions as they imagined would not seriously injure healthy lung tissue; and

\* *Brit. Med. Jour.*, Dec. 12, 1885.

† Ol. eucalypt. glob. (Platypus brand),  $\mathfrak{m}$  5 to  $\mathfrak{m}$  30, six times daily, is now my favourite remedy.

these, as one would expect, have proved too feeble to set up the necessary action in the pyogenic membrane which forms the cavity's wall.

Drainage of the cavity by incision through the chest wall appears to me to be the more rational and less dangerous method, as well as that most calculated to effect a cure.\* Almost its only danger lies in admitting air between the pleuræ; when we remember how very rare it is to come across advanced cases of phthisis in the post-mortem room where these membranes are not firmly adherent, and that this is especially so in these cases of old cavity, I think the danger of pneumothorax should not stand in the way of this procedure. There is no need of antiseptic precautions, as the cavity is already acclimatised to the external air. Once opened, the cavity can be freely and fearlessly syringed out, the bronchial opening being carefully sealed up with an aseptic plug, which also prevents the cavity's own purulent secretion from penetrating the lung. (*Vide* Bronchiectasis).

(g) Cretaceous.—I. Every now and then, in a patient who has had an attack of phthisis long past, and who has now breathing which is suspiciously cavernous, if not thoroughly so, small pellets of white matter are spat up. They vary from hard calcareous nodules to bits of pasty mortar; perhaps more usually resembling powdery dried mortar. They are spat up

\* The drawback to the successful issue of this method is the almost constant presence of mischief elsewhere. Experience has shown that this drawback is so serious that rare indeed are the cases where the procedure is justifiable.

once a day, once or twice a week, or but once in two or three months. It may be one pellet at a time, or a large mass with much coughing. Andral speaks of these as pulmonary calculi, and mentions some which were as large as nuts and beans.

II. This is a case of an old cavity which goes on secreting very slowly, perhaps a little oozing and then absolute quiescence, then a little oozing again, and so on. The moisture from the excreted portions vanishes ; is evaporated mostly, and partly re-absorbed. Finally the small excrescence becomes dry and brittle ; it falls off, and then in its new position it is perceived and promptly spat up. When the mass is considerable it is probably the contents of some angle or recess of the cavity which have gradually dried and loosened, and thus have made themselves known to the sensorium. Its occurrence gives us proof that excavation is taking place in a fibrotic district ; or it may be that advancing excavation has laid open the calcified remains of earlier disease.

III. As regards the expectoration no treatment is needed.

(*b*) Drying up.—I. The expectoration lessens and gets less purulent, for the older a cavity the more watery its contents (Lebert). A little persists in the morning with a slight cough for some time after both these have ceased in the day, and even after convalescence has been apparently well-established.

“The retrograde changes of Phthisis, whether there have been excavation or no, are usually with a copious



deposit of melanic granules ; hence abundant melanic impregnation of phthisical sputum is of good augury." The nummuli become drier and more discrete.

II. As has been before explained, every fully formed cavity takes on a pyogenic action for a longer or shorter time, but this energy generally pretty soon gets exhausted ; the patches, too, of bronchial membrane get absorbed ; the bronchi themselves calm down as they get more accustomed to their new circumstances and as the irritating discharge and, consequently, the cough lessen. In these ways then we lose the pus and mucus ; hence the wateriness of the expectoration.

III. The lesion is healing ; no special interference is needed.

(j) Contracting.—I. The most noticeable thing is the steadily increasing difficulty of expulsion as the quantity of sputum diminishes. With this difficulty the cough grows more wearing and harassing, a very trying fit resulting in the expectoration of perhaps but one nummulus.

The quality of the expectoration is much the same as in "*b*," except that the amount of watery fluid is much larger. Perhaps the expectoration most typical of a contracting cavity is made up of some four or five small, dry, pearly pellets\* floating in an ounce or more of clear watery fluid with a suspicion of froth about it ; the expectoration of the first stage plus a few pellets.

II. The mechanical conditions are constantly changing for the worse. The contraction displaces the sound

\* Hippocrates compared these pellets to "grains of hail."

portions of lung, dragging them towards itself; the solid fibrous growth and the adhesions that almost always take place further disturb and hinder the respiratory act and, consequently, the act of expulsion. We have thus the mechanical conditions of the first stage repeated, and a hacking cough is excited, which is sufficient to account for the watery expectoration. But in addition, the cavity is also secreting flaky albumen slowly, thus producing the pellets. These are of themselves resisting and not—slippery, and hence hard of expulsion; the cavity too gets irregular and devious as it contracts, the orifice probably undergoing its share, and thus other difficulties are added to those before mentioned.

Gradually, as the contraction gets more and more complete, its changes are brought about more slowly, and the lung gradually gets less sensitive to them. Finally contraction ceases, and the lung learns to take no notice of the fibrotic mass that remains, as this has now also ceased to set up any excitement by its secretion.

III. The mechanical disturbance usually produces congestion around the centre of contraction. To remove this counter-irritation is beneficial. The cough is very harassing, as in the first stage; to ease it, morphine I have found the most generally useful drug.

### **Acute Tubercular Phthisis.**

(a) **Acute Miliary Tuberculosis.**—I. The sputum is in no respects different from that of simple bronchial

catarrh. At first expectoration is absent, and there may be none throughout,\* but usually a frothy, watery, scanty sputum very soon appears ; it is spat up with great difficulty and with a constant wearing, hacking cough. It may be slightly stained with blood without any actual hæmorrhage, though even this may occasionally occur to a slight extent. Later, if there be time enough, it becomes thick and muco-purulent and may even contain the products of excavation, though dyspnœa usually carries off the patient before this stage can arrive.

II. As in the chronic first stage, so here, the deposit upsets the respiratory equilibrium, hence the cough, and therefore the expectoration. As the deposit takes place far more rapidly and is more widely distributed than in the chronic first stage, so is the equilibrium more quickly and more violently disturbed, producing the cough with corresponding quickness and making it proportionately violent. This excess of cough makes the expectoration exceed that obtaining in the chronic variety. The rapidity of deposit induces also a high state of

\* I have seen two such cases in old men, both of whom were over 60. In neither was there any marked sign or symptom referable to the lungs. I only saw one during life and he had merely slight hyper-resonance and somewhat harsh breath sounds—no dyspnœa, cough, expectoration, moist râle nor rhonchus. He simply seemed to rapidly fade away and emaciate. Post-mortem, his lungs were found thickly studded with miliary tubercle which had set up no inflammatory disturbance. In the other case there was a great deal of accompanying congestion, the liver and kidneys had one or two tubercles and the suprarenals were masses of caseous excavation, but there was no bronzing of skin or mucous membrane.

hyperæmia whence exudation into the air-sacs may also increase the expectoration and may stain it slightly with blood, causing in fact an expectoration quite similar to that which we get in the chronic first stage during the supervention of an acute attack of temporary inflammation. The lungs get so blocked with tubercle, that life becomes impossible before that first deposited has had time to break down, hence we very seldom get the sputa of excavation; but the accompanying and consequent bronchial and alveolar catarrh may reach the muco-purulent stage and hence muco-purulent expectoration may arise late on in the disease.

III. The cough has to be soothed in the same way as has been indicated in the irritative cough of the chronic first stage.

**(b) Acute Catarrhal, or Pneumonic, Phthisis.—**

I. There may be no expectoration at all, or merely a slight, watery, irritative one, as in “*a*:” this however is very rare.

Usually the irritative sputum occurs at the commencement; in a month or less the mucus becomes thick, begins to be mixed with pus cells, and rapidly acquires muco-purulency. This muco-purulent expectoration is often in large quantity, and comes up very easily, the amount being fairly constant throughout the twenty-four hours. When it is much it is commonly diffuent and is rarely stringy from admixture with tenacious mucus. It may get somewhat concocted and come up in large slimy gobbets. Prune-juice sputa occasionally occur (as in a case I saw at Brompton); some say they are

especially frequent, but Walshe considers this to be a grave error. In the spittoon there is sometimes a thick head of froth. Trousseau tells us that in this phthisis galopante, the expectoration is not to be relied on in young children, as in them it has no constant character. Late on in excavation, if the patient live, the expectoration becomes less in quantity, more bronchitic, and there is more difficulty in its expulsion.

II. Dr. Pollock mentions a boy of thirteen who died with cavernous gurgle in six months, and yet there was no expectoration. This I cannot explain, unless the secretion were swallowed instead of being expelled, as in younger children, but then you would have expected some sign of its presence either in diarrhœa or vomiting. Dr. Law states the case of a boy of five, who died on the tenth day without there having been any expectoration. Here both lungs were in a condition of pneumonia (hyperæmic to red hepatisation stage) with small scattered tubercles especially near the bases. In this instance the quickness of death forestalled expectoration.\* The irritative sputum is due

\* I had charge of a similar case at Brompton under Dr. C. T. Williams. A diminutive girl of fifteen had never been well since an attack of "typhus," six months previously. Had had throughout occasional heavy night sweats; there had been much wasting, and for two months she had been confined to bed. She had never had, nor did she up till death have, any cough or expectoration. On admission there were great lividity and dyspnœa, herpes labialis and coarse crepitations in both lungs. She died in a week. Much 2nd to 3rd stage pneumonia was found in both lungs post mortem, with small scattered nodules of softening identical in appearance with yellow tubercle, whilst in the right lung were patches of pigmented miliary tubercle which had probably existed some months.



to the disturbing deposit as in “*a*”; but here it is commoner and in larger quantity, because the alveolar catarrh tends to involve the minute bronchi and always affects the endothelium of the alveoli, whereas the interstitial tubercle produces a morbid secretion only by indirect irritation.

Excavation usually begins from the fourteenth to twentieth day. The muco-purulent expectoration arises from the rapid breaking down of the softened deposit, both that blocking the alveoli and the interstitial infiltration. The process being very rapid, there is no tendency for the disintegrated products to cling to the tissues (compare the clean surface of a freely discharging ulcer with the shreddy coating of lymph which sticks about a more sluggish one): these products too break completely down into pus (unless indeed fragments of tissue are spat up whole); thus there are but few of the disorganised cells and other *débris* which are drier, more irregular and rough in outline, and hence do not form so mobile a secretion as the round pus cells—for these reasons cough is but little needed and, therefore, but little mucus (the product of bronchial irritation) is mixed with the sputum.

When it takes the form of gobbets or semi-nummulated lumps, it still comes up easily, as these are very smooth and slippery, consisting chiefly of pus cells entangled in mucus. These are found later on when cavities have formed, and the secretion is able to lie some time quietly before the patient is prompted to expel it. They are of course generally mixed with the

purulent or muco-purulent expectoration, coming from other portions of the lungs.

Finally the expectoration lessens, partly because the general vitality is waning, and partly because the lungs are losing their elasticity, and therefore lend themselves less easily to expulsive efforts. It becomes more bronchitic because the tubes are gradually sharing in the general inflammation.

III. As regards the expectoration no special treatment is required except towards the end, when ether and ammonia are of great value in aiding its expulsion, and in this way preventing fœtor.

### **Hæmoptoic Phthisis.\***

I. The first intimation is usually hæmoptysis, one teaspoonful to several ounces ; then perhaps the next is another attack of hæmoptysis, which may be no more or much more in amount. Usually there are several of these attacks with no after-symptoms, except maybe the expectoration of a little mucus, which for the first two or three hours is perhaps slightly stained. But sooner or later the hæmorrhage is followed by a small expectoration of consistently stained, thick, mucoid sputum, the staining lasting probably three or four days. There are sometimes repeated supplementary

\* The existence of this condition, apart from bacillary infection, is not generally admitted. It is undoubtedly rare—I have only seen the one case recorded below—and I am inclined to think this rarity accounts for much of the scepticism concerning it.

attacks which keep up the staining for a considerable period. The cough is generally slight and not trying, and the expectoration does not necessarily increase in amount as the disease progresses ; in fact, towards the last, expectoration may be scanty, difficult and suffocating, though the sputum itself lie loose enough in the tubes. One is often surprised by the sudden expectoration of dark clotted blood—the blood of hæmoptoic phthisis being commonly bright and arterial—this having lain for a long time in the lung, till indeed it may have become fœtid.

During the first stage it is important to diagnose this disease from the morning expectoration of a teaspoonful or so of blood, which is rather a common ailment in young women, and is related to hysteria. The diagnosis is usually easy : in this latter the blood is venous and diluted, not frothy, only spat up before breakfast, never much more than a teaspoonful, has a characteristic fœtidity, and the after-expectoration, if it exist, is devoid of staining. This persistent staining of the sputum is of itself sufficient to clench its origin.

These hæmoptyses continue at about the same intervals and in about the same amounts till death : the period of excavation is liable to sudden, profuse, and even mortal attacks as in any other variety of phthisis, but the characteristic hæmoptoic bleeding continues unchanged.

If excavation ensue, one gets its ordinary expectoration complicated with the *débris* of the effused blood ; this consists chiefly of fibrin, varying in colour from

brown to almost white, according to the age of the nodule.

II. The patient is suffering from hæmorrhagic diathesis, the portion of the body affected, always chiefly and often solely, being the lungs. Hence, on any exertion, or at any period when they are unusually turgescient—from plethora, hepatic congestion, puberty, menstrual epochs, &c.—the lung capillaries are apt to allow blood to ooze from their unprotected sides into the air-sacs. It is then perceived and, if in small amount, is mostly coughed up, though some no doubt is always left in the air-sac, and is either re-absorbed or becomes converted into a blood-nodule. If the amount is in any way large, or the first expulsive effort weak, then a great deal of the blood is merely moved from the air-sac into the bronchi and the following deep-drawn inspiration sucks it down into other air-sacs, or into the bronchioles which supply these. Thus, as the hæmorrhages go on more and more of the lung becomes blocked. It is because of the solidity consequent on this that the expectoration late on becomes difficult and suffocating. This blocking also throws more work on the free portions of lung, which get congested, and therefore more liable to hæmorrhage.

The blood in its long journey to the larynx has to pass over great lengths of bronchial tubing; if these contain sputum, it necessarily becomes stained. Besides, the mere presence of blood in, and passage through, the tubes may excite them to secretion, as it previously excited the air-cell to expel it. Hence, in

this way again we have staining. After many repetitions of the attacks the tubes become chronically irritated, and a continuous mucoid sputum results. Effused blood, unlike pneumonic deposits and tubercular nodules, is exceedingly well borne by the lung; hence the slight cough. Should any blood be drawn into any air-sacs in a pneumonic state, it naturally acts as a fresh excitor and hastens the expectoration of excavation. But in healthy air-sacs it may lie innocuous for an indefinite period, and take unto itself a thick fibrous capsule, till finally, perhaps, it is spat up as a fibrinous colourless mass by the bursting of that portion of the capsule nearest to the bronchiole. Or it may be similarly spat up when it has only had sufficient time to clot and become dark, or stay rather longer till the yellow—hæmatin—period be reached. It is this unirritating nature of the blood which accounts for the remarkable absence of excavation and its expectoration in this form of phthisis: the fatal result being often reached by dyspnœa, as in fibroid phthisis, or by hæmorrhage.

III. This is a rare and very hopeless form of phthisis. Treatment should be chiefly directed to ridding the lungs of every drop of exuded blood. The patient should be encouraged to cough it up as much as possible. Sedatives should therefore be avoided. Hæmostatics should be persisted in—*e.g.*, lengthened courses of ergot or perchloride of iron.\* Menstruation should be established, if absent, and the bowels kept freely open.

\* I should now have more faith in calcium chloride.



The following is a brief abstract of a typical case of which I had charge under Dr. Reginald Thompson : Jane T., 21 ; father died of hæmoptysis ; mother of phthisis. One brother and one sister suffer from hæmoptysis. In September 1880, she brought up a quarter of a pint of blood and a similar quantity in March 1881. Catamenia regular till July 1881, then ceased for three periods ; on their return there was fresh hæmoptysis. Admitted into Brompton, November 1881 ; cheerful, well-nourished, but rather pasty-looking. Slight cough, with stained sputa. Moist clicks and increased vocal resonance heard at right supra-scapular fossa ; left apex dull, with moist, coarse crepitations, both anteriorly and posteriorly ; fine crepitations at left posterior base. She died March 30, 1882. During her stay at Brompton she had hæmoptyses at intervals of one to five weeks, usually about five ounces in amount, but varying from one to ten. Her breathing got steadily worse, and for the last month she had orthopnœa ; she died from gradual asphyxia. Her lungs were filled with nodules of blood of various ages, and there were a few small cavities from which similar nodules had evidently gone, as if they had been scooped out, the walls being smooth, rounded, and having the yellow stain of hæmatin. No tubercles were found,\* nor was there any marked emaciation, notwithstanding the loss of blood and the fact that she had been persistently kept on a spare diet.

\* There was no examination for the bacillus.

### Bronchiectasis.

I. The expectoration is profuse, contains usually no elastic tissue, but consists of creamy, diffiuent pus, mixed, however, with some bronchial mucus of a greenish hue, though this latter is generally in small proportion. It comes up easily and runs out of the mouth with practically no coughing. As there is usually a large residuum of expectoration in the tubes any movement is liable to induce the removal of a portion of this, and thus cause a sudden choking deluge, resembling the expectoration of an empyema opening through the lung ; but the expectoration of bronchiectasis differs from that of empyema in being much easier, with little or no coughing, and in there being also a continuous discharge between the sudden outbursts. The amount is large, a pint in the twenty-four hours being by no means remarkable. It has a characteristic sweetish sickening odour, which Guttman compares to the smell of a soap manufactory. This is extremely penetrating, so that antiseptic inhalations, &c., have but little power over it. In the foulest shreds of sputum are found long, slender, colourless needles, lanceolate and usually straight, single or in tufts, consisting of a combination of palmitic and stearic acids (Guttman, who says they are found in most *gangrenous* discharges). In the putrid stage *gangrenous cores* are also found.

“I have never known hæmorrhage to any notable amount produced by chronic bronchitis with dilatation

alone ; if hæmorrhage exist, and there be no evidence of mitral disease, the inference that the excavation is tuberculous becomes matter of necessity." (Walshe.)

The sputum on standing divides into three layers—  
(1) An upper transparent and very fluid one. (2) A middle one consisting chiefly of flocculent mucus. (3) A lower, which is opaque and almost exclusively purulent. Hæmatoidin crystals may be found in it. (Guttmann.)

II. It is profuse because the extent of surface diseased is great, many tubes of both lungs being generally involved sooner or later, some of which are dilated to large fusiform cavities ; also because mucous membranes are naturally secreting surfaces. In a well-marked case there is but little mucus ; the mucous membrane is too diseased to secrete anything but pus. Such tubes as are not affected remain almost perfectly healthy, as there is but little harassing cough : hence, though these secrete mucus, they do so in small amount. Finally, Biesner, quoted by R. D. Powell, gives atrophy of mucous membrane as one of the causes of bronchiectasis, thus additionally explaining the comparative absence of mucus.

The ease of expectoration is due partly to the extremely purulent nature of the sputum, the absence of nummuli and rough jagged *débris*, &c. ; partly because it has not to be forced out of an irregular cavity through a small hole at an angular disadvantage, but is already in a too spacious bronchus ; but chiefly because the tubes have lost most of their sensitiveness,

allowing the pus to fill or even distend them, the overflow only being spat up, and even running from the mouth of its own accord, should the patient be lying with his head low. The expulsion is fairly constant; there is no reason why it should not be so if the secretion be, for the outward passage is patent, free and slippery.

As to the offensive odour I again quote from Dr. Ewart's Gulstonian Lectures: "Oxygen does not seem to decompose offensively true phthisical cavities because they have but little mucin; and chemical metamorphosis has taken place in the dry state, at the inner surface of the capsule, and its products have reached so low a standard as to afford little room for further degeneration. But offensive decomposition is characteristic of bronchiectasis; for oxygen acts here on bodies of complex organisation in the moist state; the decomposing mass largely consists of mucin, which is a substance in its moist form usually prone to decay; fresh pabulum, too, is being constantly added: hence we often get mucous fermentation and acid reaction in bronchiectatic sputa." (This does not contradict the fact that in bronchiectasis tubes secrete but little mucin, and that the expectoration in tubercular phthisis is richer in mucus than that of bronchiectasis, for in the former the vomica where the actual phthisical *secretion* occurs contains practically no mucin, and it is only during its *expulsion* along the irritated tubes that the admixture takes place.)

Guttmann evidently looks upon the needles of

palmitic and stearic acids to be the chief source of the fœtor. Others assert that margarin and butyric ether are to be found in the sputa. One would not expect hæmorrhage ; in so chronic an inflammation fibrosis is always certain to keep ahead of excavation. At first the vessels are well protected by the stout epithelium and later on by this fibrosis.

III. It is extremely necessary here to insist upon the complete expulsion of the sputum ; if swallowed, it is peculiarly liable to upset the digestive organs, and its long continuance in a thin-walled sacculus may produce pyæmia by absorption.\* The spittoon and room should be kept disinfected, but with the utmost endeavours in this direction it may be impossible to remain for long in the same room as the patient. Antiseptic inhalations are usually worthless. The internal administration of iodoform in large quantities—up to 25 or 30 grains daily, as recommended by Dr. Shingleton Smith—seems well worth a trial. Also the use of such drugs as alum, tar,† and terebene, in order to lessen the secretion should not be neglected. In a well-marked case at Brompton, under Dr. Williams's care, the overpowering

\* Persistent attempts should be made by adopting various positions with the head lowered (especially lying semi-prone on the more affected side upon a couch with the head and neck hanging down over the edge), or by the manipulation for artificial respiration, particularly the forcible compression of the chest laterally during respiration.

† In one of my private cases tar pills taken to the extent of 30 grains daily for some months proved very beneficial in lessening fœtor and expectoration, but this patient finally developed an ischio-rectal abscess and died of chronic septicæmia.



fœtor was almost entirely removed on the largest and most superficial, but not the lowest, dilatation being drained and washed out through the chest wall, thus showing that the fœtid decomposition may be taking place in one locality only, and is probably the result of purely mechanical conditions.

### **Fibroid Phthisis.\***

I. The most characteristic thing about the expectoration is the great difficulty of its expulsion, and the steady increase of this difficulty. The cough is consequently very wearing, causing retching and vomiting. At first the sputum is watery and often stained, as in the first stage of ordinary phthisis with occasional slight hæmoptysis. Later it becomes muco-purulent and may be abundant.

II. The fibrosis accounts for the difficulty in a manner previously explained. Muco-purulency and

\* Under this heading I include all those cases where cirrhosis of lung is the predominating feature; it does not exclude the co-existence of tubercle, catarrhal processes, or dilated bronchi; clinically indeed there is a strong tendency for these cases to merge into bronchiectasis, but still typical cases are easily distinguishable and causally the difference seems to me essential, the one being a disease primarily of the bronchi, the other of the interstitial parenchyma; if, as Hilton Fagge suggested ("Principles and Practice of Medicine," vol. i. p. 905), nearly all cases of fibroid phthisis are tubercular the causal difference becomes even more marked. Pathologically, too, the distinction holds good, for Bastian states (Reynolds' System) that 6 out of 30 cases of fibroid phthisis had no dilatation of tubes, and Hilton Fagge asserts that bronchiectasis may occur "without any change in the pulmonary tissue, unless it is perhaps emphysema." But see "Fibroid Diseases of the Lung," by Clark, Hadley and Chaplin.

abundance are due chiefly to the production of cavities and dilated tubes.

III. To soothe the irritated lung and so lessen the cough, is the chief thing to be done. Treatment for this purpose has already been indicated.

**Varieties.** (1) Carbonaceous phthisis or miners' lung.—The peculiarity of the expectoration here is its impregnation with carbon. Dr. McKellar, in one case, collected weekly, for some weeks, two ounces of carbon which he obtained by precipitation from the sputum. This carbon comes from the lamp-black given off by the miners' lamps. When once there has been free inhalation, the lungs become producers of carbon, continuing to expel it for years after the patient's removal from the original cause. McKellar states he never found tubercle in these lungs, though the patients might have had phthisical parents.

(2) Grinders' rot, potters' phthisis, &c.—In the expectoration are found grey particles of stone grit or of metallic substances, these having been previously inhaled as they flew off from the grindstone or from the instruments ground upon this.

(3) From lesion of the heart.—Here, as the fibrosis is due to stagnation of blood, there is naturally a greater tendency to stained sputa and to slight hæmoptyses.

(4) From pleurisy, especially purulent pleurisy.

(5) The other forms are due to intrinsic disease of the lungs themselves. These are by far the most numerous, but their sputa does not help one to diagnose them from one another, except by the discovery of specific germs.

**Emphysema.**

I. The habitual sputa, when emphysema occurs independently of bronchitis, are frothy, liquid, muco-epithelial or watery, and never sanguineous ; they may consist of the pearly sputum of Laennec's dry catarrh. (Walshe.)

II. The following extract from Dr. Ewart's *Gulstonian Lectures* (1882) shows its right to be classed as a lung phthisis, and also explains why it has the sputa mentioned above :—"Its bullæ, from the breaking down of air-cells, are often so big as to be 'cavities.' Should they be surrounded by thickened pleura, their bronchus be obliterated, their gaseous contents become fluid, you get a 'cavity' which only differs from the ordinary ones in the limpidity of its contents and the polish of its walls."

III. None required when bronchitis is absent.

**Morbid Growths.**

I. The early expectoration common to all is the same as that occurring in the first stage of chronic tubercular phthisis.

(1) **Hydatid.**—Expectoration, at first catarrhal ; later sanguineous, and then come fragments of cyst-wall and hooklets : it is usually paroxysmal, and may always be preceded by suffocative dyspnœa with great general anxiety : hooklets may continue to be spat up for months.\* If it come from the liver, then you have

\* In a case under my care for two years there were also frequent hæmoptyses, usually not more than a drachm or so, but occasionally reaching several ounces.

paroxysmal cough, with copious expectoration of bile-stained hydatids, which are perfect or imperfect, and may or may not be mixed with a mahogany foetid fluid, or sometimes even with visible shreds of sloughed lung. (Walshe.)

(2) **Cancer.**—Cough is invariable; and it is very seldom dry. The expectoration may be simply catarrhal, purulent, or sanguineous. In this last the blood seems thoroughly mixed with serosity, mucus, or muco-pus, and thus the usually opaque or perhaps slightly transparent sputa become pink, or the colour of red (or black) currant jelly. Recognisable encephaloid detritus has rarely been found in the expectoration: occasionally it contains some cancer cells; occasionally it is excessively foetid. Hæmorrhage in large amounts usually occurs. (Walshe.)\*

(3) **Syphilis.**—Nothing diagnostic in the expectoration. The phthisis is usually of the fibroid variety, but gummata occur, and may lead to excavation.

(4) **Ova of Distoma Ringeri.**—Irregular, intermittent hæmorrhage, with slight cough, with less active periods during which small pellets of viscid brownish mucus are spat up several times a day. Considerable hæmorrhage or expectoration is easily induced by coughing, &c. The sputum contains the ova.

II. and III. call for no special remark.

\* Cancer may bring death previous to any tissue destruction, as in a case lately under my care: then the expectoration is that of bronchitic asthma.

**Lung Abscess.\***

I. At first there is the expectoration of very acute lobar pneumonia—viz., scanty, difficult, watery, rusty, or none at all, according to the irritation the growing abscess produces. Then comes a sudden, purulent, bloody, chocolate-coloured expectoration which is thick or liquid. “It is the quantity of the expectoration, this quantity suddenly increased, its peculiar character, its fluidity following on viscidty, which guides us in diagnosing the bursting of an abscess into the bronchi.” (Trousseau.) The expectoration differs from that coming from a phthisical vomica (tubercular) in containing none of those friable fragments of softened tubercle mixed up with the purulent expectoration, and also in having no elastic tissue or bacilli. Later on, it is true, it will contain these, should it lead to

\* By this I mean the single acute abscess which occasionally complicates lobar pneumonia; this must be carefully distinguished from those multiple and irregular foci of suppuration which result from pyæmia or lobular pneumonia. It is very rare, and some authors (Hilton Fagge, *e.g.*) doubt that it ever occurs, but this doubt seems to me hardly justifiable in the face of such clear and masterly records of cases as Trousseau gives us in his “Clinique Medicale” (5th edition, vol. i. p. 856) from which I have chiefly drawn the following description. I lately took charge of a case of lobar pneumonia for Dr. Foster, where I diagnosed abscess, and should have had it opened through the chest wall, had not the man’s general condition been too hopeless; post mortem, this diagnosis was confirmed. I have since seen one other in consultation where my diagnosis was also confirmed post mortem. The pneumonia was in the prune-juice stage: there was no sudden discharge, but the expectoration was fœtid and grumous and the odour of the breath distinctive.



breaking down of lung parenchyma : but the difference to be noted is that the tubercular vomica is far the likeliest to have it, and to have it in greatest quantity at first, when it is full of broken-down tissue, whereas the true abscess cavity is pretty certain to have none at first, but may contain a little afterwards. When the acute stage is past it is impossible to tell an abscess cavity from a simple secreting cavity of tubercular phthisis by the expectoration alone, for this may be identical. Dr. Douglas Powell states that the expectoration from an abscess formed in the course of a lobar pneumonia is often fœtid at first, and always becomes so in a short time.

II. It is a local acute inflammation going on to suppuration as in any other part of the body. The surrounding pneumonia naturally produces ordinary pneumonic sputum. Then comes bursting with evacuation of abscess contents and a lessening of the tension and surrounding inflammation. Now, if the abscess heal, the purulent expectoration gets non-fœtid and gradually ceases ; if it become tubercular, you get the expectoration of the excavation stage of chronic phthisis, or it may continue as a simple pus-secreting cavity.

III. Should the patient's strength admit of it, I have no doubt of the advisability of draining the abscess through the chest-wall. This should be done even after the abscess has opened into the bronchus, unless the cavity appear to be rapidly closing of its own accord.

**Apoplectic Phthisis.**

I. "The only symptom really important is hæmoptysis in forms of tinged mucus, striæ of blood, pure blood, rarely florid, but rather dark and of a bistre tint. The quantity is habitually small. I have never once seen profuse hæmoptysis attendant upon nodular apoplexy. I have never seen bistre or sooty-looking hæmoptysis except in cases of this sort. Where it has ceased before death the nodules have shown signs of absorption. Red discs may often be found in sputa of persons with mitral disease where no blood is visible to the naked eye and, conversely, numerous nodules may be found post mortem, though not the slightest hæmoptysis had existed during life." (Walshe.)

II. It is always venous blood, and hence dark. It often, no doubt, lies a long time in the inter-alveolar tissue before being expectorated, and thus gets still darker from destruction of its red discs. Sooner or later the effusion will probably irritate the lung, and bring about the ordinary expectoration of excavation or fibroid phthisis; or it may occur rapidly, as in the case of pyæmic embola.

III. The expectoration is only a symptom of a lesion of some organ other than the lung, to which treatment must be directed.

**Pleuro-Pneumonic Abscess.**

(*Empyema, primary or secondary, opening through the lung.*)

I. Trousseau says the expectoration is at first bloody and mucous, later the colour of apricot marmalade. This, of course, refers to that kind which begins as an empyema pure and simple, opening at last into a bronchus; where the empyema is the result of a vomica bursting through the pleura, then the previous expectoration might be any one of the varieties occurring in chronic phthisis.

To take Trousseau's case first. Here the quality of the discharge and the manner of its onset give nothing by which it may be distinguished from an abscess in the lung parenchyma (*vide* Lung Abscess.) But there are the following important diagnostic points, speaking with reference to the expectoration only:—(1) Trousseau says that, if it be in the tender lung parenchyma, it never opens into the bronchus later than the twentieth to twenty-fifth day, that this is equally the case whether it have previously opened into the pleural cavity or do so afterwards; whereas abscesses opening on the fortieth to sixtieth day are either pleural or between the lobes, always excepting those of children and puerperal women, which, though pleural, may burst into a bronchus on the fifteenth to twentieth. (2) Trousseau further states that a parenchymatous abscess cannot contain a litre, while a pleural one may hold as much as four perhaps; hence the importance of measuring the first discharge. (3) And

perhaps most diagnostically, a pleural abscess will, as every one knows, continue secreting rapidly in large and increasing quantities. In a case of Trousseau's five litres were brought up the first night and, in less than a week, eleven; while Legroux tells of a patient who brought up forty-three litres in a comparatively very short time. Now a parenchymatous abscess, as a rule, tends to secrete less and less, and at no time in very large quantity. (4) Naturally there will have been less irritation of lung and bronchi previous to bursting; the preliminary expectoration, therefore, will not be so much as with the lung abscess, nor probably at all bloodstained. (5) The breath will probably be more foetid, and will remain foetid longer, in the pleural abscess. (6) The discharge too, is more variable; it is liable to come in big gushes, nearly choking the patient, out of both mouth and nostrils at once, like a vomit.

When a vomica of tubercular phthisis breaks through into the pleural cavity there is the previous history of excavatory expectoration, then suddenly, with the pneumothorax comes an extremely painful and wearing cough bringing up an expectoration containing serum and mucus added to the products of tubercular excavation. In a few days, if the patient survive, the sensitiveness and cough lessen while the expectoration becomes more and more purulent, creamy and homogeneous. Here there is no sudden discharge of pus in great quantity, but the amount begins at a minimum and gradually increases.

The fistulous empyema, once established, its mode of origin is unimportant in so far as concerns the expectoration, the character of this being the same in both cases, viz.:—(1) Brought up very easily with little or no coughing. (2) Usually in scanty amount or absent, except two or three times in the day when it is sudden and very copious, sometimes rushing out of both mouth and nostrils like a deluge, in fits easily excited by unwonted change of attitude, tickling cough, or sudden exertion ; its method of expulsion being then very often like that of a violent hæmorrhage. (3) Usually it is a creamy diffuent pus containing no elastic tissue (unless this has previously existed or excavation be already induced, which in any case usually soon comes). (4) Its total daily amount is always large, and yet causes comparatively little exhaustion. (5) Its odour may be that of healthy pus, or extremely fœtid. (6) Its colour is unrelated to odour and is usually that of normal pus, though a green hue is not rare and does not assert any connection with the liver. (7) It may contain crystals of hæmatoidin and cholesterin.

II. It is easy because the diaphragm, ribs, &c., have free range of action so long as any fluid exist in the pleura, and because the channel of exit is usually free and large. It is true that this has sometimes a valvular entrance, but even then the mechanical conditions are generally either a case of no exit at all or else a free one. Should the collapse of lung be great, with much displacement of heart, then there will be a residuum of pus unable to be spat up, though the compensating



falling-in of the side will gradually decrease the amount of this ; but when there is this residuum still the expectoration of the tidal pus will be easy. Its expulsion is apt to be fitful because there is a considerable cavity accustomed to the contact of the pus ; this therefore fills before any attempt is made to expel it. Should, however, any exertion, &c., suddenly or violently alter the relation of the pus to its surroundings, these resent this change and bring about its expectoration by a reflex act. Especially is this the case if the exit be valvular—*e.g.*, a sinuous tract which in some positions is patent, and in others devoid of continuity of lumen.

Practically the condition is one of open empyema ; the secretion is therefore pus. Elastic tissue seldom exists, for the tendency of the pyo-pneumothorax is rather to arrest than hurry on excavation, the lung becoming collapsed and fibrotic. The amount of expectoration is large, because the extent of surface involved is large, and because that surface is naturally a secreting surface. Fœtidity, if present, would be due to damming up, with its attendant imperfect oxidation. The painful cough and constant watery expectoration which occurs when it is the result of the giving way of a phthisical excavation are due to the intense pleurisy set up by the entrance of air and some lung detritus into the sensitive, healthy, pleural cavity.

III. As a rule, no time should be lost in giving a free exit to the pus through the chest wall. In children I should wait a day or two, as I have seen two or three cases where the first outburst was also the last,

and was followed by speedy return to health, just as in other instances of empyema in children there is no return of the pus after a single aspiration.

### Gangrene.

I. Guttman says the sputa are very abundant, somewhat fluid, and of a dirty greenish-yellow colour. As with the bronchiectatic sputa, so here he finds three strata appear on standing :—(1) An upper greenish-yellow, opaque and frothy. (2) The next is very transparent, albuminous, and almost serous. (3) The lowest is yellow and opaque, consisting almost entirely of pus-cells with a detritus containing many dull yellowish-white cores, varying in size from a millet seed to a bean, which are very foetid and possess fatty crystals. Traube states that these cores differ at different stages of the disease :—At first they consist of pus-cells and detritus, then become a dirty grey with fat globules in the detritus ; next they are chiefly made up of detritus with fat globules and crystals ; and, lastly, become much more numerous, and are grouped in bundles. Trousseau says the foetid odour is nearly, if not always, pathognomonic, though it is sometimes absent at the beginning and end (if there be a tendency to cure) of the disease ; that the appearance and colour often in no way differ from the muco-purulent expectoration of catarrh ; that the sputum of bronchiectasis differs from it in not having exactly a gangrenous odour, in being always abundant, and in being nearly entirely made up of foetid gangrenous mucus (*sic*), while in true gangrene

there is usually a peculiar appearance of animal detritus. Guttman finds no elastic fibres, or only for a very short time and in small quantity.

Walshe speaks of four varieties : (1) Diffuse. Expectorations profuse, frothy, and purulent with gangrenous odour. Power to expectorate being soon lost. (2) Circumscribed. The previous muco-purulent expectoration (which is rarely blood-stained in adults, but often so in children), so soon as communication is made between a bronchus and the gangrenous tissue, becomes dirty-greenish, yellowish-brown, or ashy-grey ; at the same time it becomes very liquid with an odour which is gangrenous, resembles that of wet mortar, or at any rate is very fœtid. This fœtor of the breath is three times as common in adults as in children, and in them it may vanish for some minutes, and then suddenly become again intolerably fœtid, though there has been no interposition of anything like cough, &c. (3) A gangrenous cavity may get pyogenic or pseudo-mucous, the expectoration having no gangrenous odour. (In a case which he mentions the sputa became putrid temporarily from time to time.) (4) Cruveilhier describes an odourless form ; the texture of the lung falling into shreds, and there being no smell during life or after death. (Dry gangrene.) Trousseau remarks that the odour and a similar expectoration may occur in pleuro-pneumonic abscesses which are circumscribed, especially interlobar ones, for in these the pus may be in small quantity and horribly fœtid.

III. Disinfection is the chief thing to aim at ; if the

disease be localised, the question of incising and scooping out the gangrenous portion should be considered.

### **Actinomycosis.**

I. The sputa, after the stage of initial irritation, become purulent; but the pus is thin, viscous, and somewhat tenacious, containing small nodules of a grey or yellow colour about the size of a poppy-seed, which under the microscope exhibit all the characteristic features of the ray fungus.

(Drs. C. T. Williams, Douglas Powell, Tatham and Reginald Thompson, the physicians under whom it was my happy lot to work at Brompton, I most sincerely thank for their kind permission to make use of their cases.)

### III. ON CATARRH

#### (A) ON THE NATURE OF ACUTE PNEUMONIA IN CHILDREN

THE plan of inquiry adopted by the Collective Investigation Committee on "Acute Pneumonia" prevented prominence being given to differences of type in different localities; my fortune as house-physician at the Pendlebury Children's Hospital, Manchester, at St. Thomas's and at the General Hospital in Birmingham, has given me special facilities for observation of the disease from this point of view.

The statistics\* I am about to submit to you are based on an examination of thirty-nine cases at Manchester and Birmingham respectively, and of nineteen in London. My numbers are limited, because in many instances I found the notes wanting in one or more particulars. In justice to Pendlebury, I must say that this deficiency was not found in the notes of that hospital, which are very complete, but in my own notes taken at the General Hospital. The London cases are few, because we had very little room at St. Thomas's to

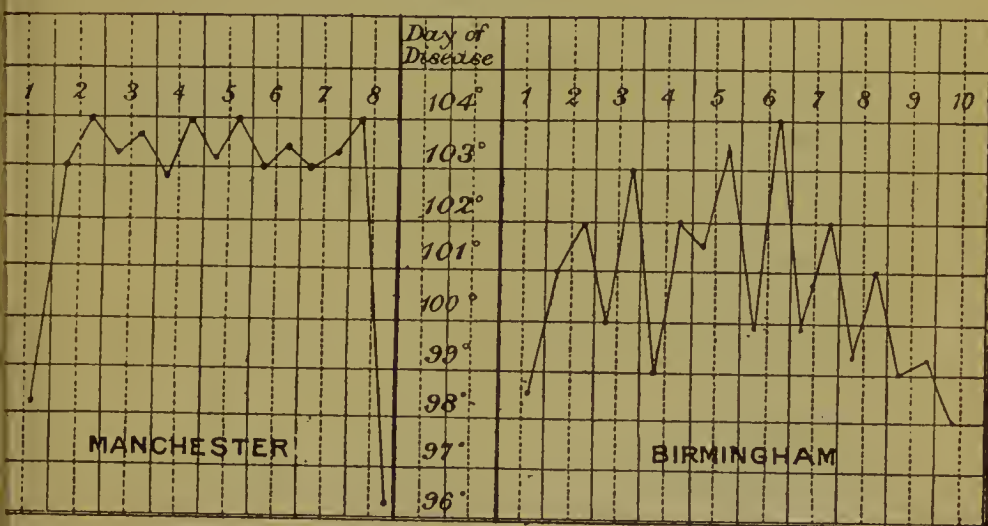
\* I have to thank the physicians of the General Hospital, Dr. Stone of St. Thomas's and Drs. Ashby and Hutton of Pendlebury, for their generous permission to make use of their cases.



devote to the medical diseases of children.\* I shall refer to these London cases only incidentally, I have inserted them because they form a connecting link between the extremes of Manchester and Birmingham.

The difference which is the first to strike one lies in the march of the temperature. My impression was that

FIG. 1.



this would be represented by curves like those in Fig. 1—the Manchester temperature having a higher range, holding this more steadily, and ending with a sharp crisis; at Birmingham some of the daily maxima being equal to those of Manchester, but the minima much lower; that is to say, the daily variation (which may amount to hectic) being much greater, whilst the fever ends either with a prolonged crisis or with a lysis.

\* Except in a single instance this paper deals entirely with pneumonia occurring in children of not more than fourteen years.

Statistics show, however (*vide* Table I.), that this impression was not correct. The daily variation in Manchester was  $2^{\circ}$ , whilst in London and Birmingham it was only  $1.5^{\circ}$ . Reference to two typical cases shown in Fig. 2 will explain this mistake. The great variations in Birmingham temperatures occur not daily, but extend over groups of three or four days; hence these do not appear in averages struck from the diurnal

TABLE I.

Nature of the Pyrexia.	Manchester.	London.	Birmingham.
Average procrisial maximum } temperature . . . . }	104	102.8	101.7
Average procrisial minimum } temperature . . . . }	102	101.3	100.2
Crisis occurred in . . .	34	15	14
Lysis occurred in . . . .	4	4	18

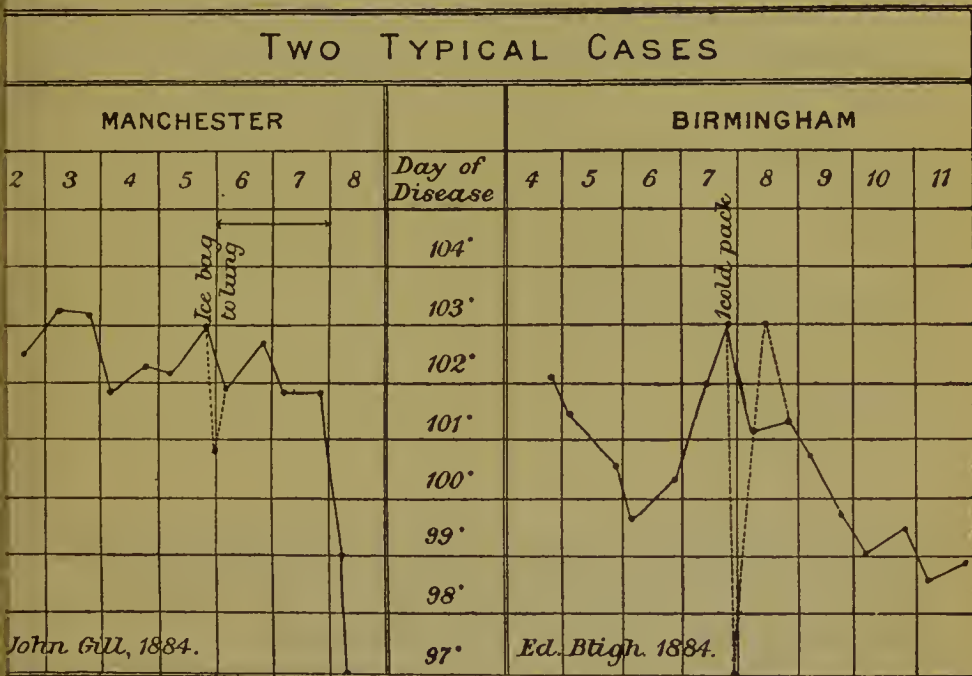
variations alone. As to the higher range of the Manchester temperatures, that comes out fairly well,  $2.3^{\circ}$ , though this is a good deal lessened by the almost universal and persistent employment of antipyretics at Manchester, whereas at Birmingham these were very seldom used, the treatment indeed usually tending to stimulation.\*

The different ending is also well marked, but what is

\* When antipyretics were used at Birmingham they acted much more powerfully than at Manchester. This is well shown in

not indicated is the length of time occupied by the crisis. By crisis I have understood for statistical purposes a steady unbroken fall to normal, lasting not more than twenty-four hours; this definition enabled me to include fourteen of the Birmingham cases under this heading, but in many of these the fall took between sixteen and

FIG. 2.



twenty-four hours, whilst in most of the Manchester ones its duration was less than twelve.

As to the lung lesion the difference seems of deeper import. The statistics (Table II.) do not sufficiently exemplify it, as the consolidation in this district is

Fig. 2, especially if we remember that, according to Dr. Hutton, the local application of ice to the affected part is a more effectual antipyretic than a cold pack.

merely in the foci of an inflammation of moderate intensity but wide area, usually indeed affecting both lungs; at Manchester inflammation is more localised but of much higher grade, and solidifies all the tissue

TABLE II.

Lung Lesion.	Manchester.	London.	Birmingham.
Consolidation present in } both lungs . . . . . }	5	7	22
Consolidation present in one } only . . . . . }	30	10	5
Catarrh, but no consolidation .	0	2	12
General pulmonary catarrh } as well as consolidation . }	3	3	7
No signs . . . . .	4	0	0

which it attacks. Here, in fact, we have the lobular hepatisation of Rilliet and Barthez; at Manchester the disease is a true fibrinous lobar pneumonia.

Table II. refers to the state of the lungs on admission only; but as, both at Manchester and here, this occurred on the average on the fourth day, the statistics should be relatively correct.

Next, I will take the frequency of the gastro-intestinal complication (Table III.). For a case to be classed under this heading persistent abdominal pain, vomiting, irritative diarrhœa, or constipation must have existed throughout the period previous to admission (A), or

during the whole pyrexial period subsequent to this (B), or, as was most frequently the case, from the onset to the crisis. The vomiting and constipation usual at the

TABLE III.

Gastro-Intestinal Complications.	Manchester.	London.	Birmingham.
(A) Noted in the history . .	3	6	13
(B) Noted after admission . .	4 out of 21	5	12

onset, as well as the loose stools which are common at the crisis, have been carefully excluded.

These statistics represent the real state of things as regards Manchester and London, but they are far from showing the great importance of this complication in

TABLE IV.

Mortality.	Manchester.	London.	Birmingham.
Fatal cases . . . . .	1	0	4

Birmingham. For here, when gastro-intestinal mischief exists, it nearly always takes the first place in the clinical picture and demands all our therapeutic attention: whereas in Manchester and London the lung lesion was as a rule our chief care, the intestinal catarrh being of slight severity.

Fourthly, as to mortality (Table IV.), the numbers



are far too small to deduce any valuable conclusion from them, but they are inserted because they bear out my own strong impression of the far heavier mortality attendant on the Birmingham fever. This is chiefly due to the gastro-intestinal complication; such cases being always the most anxious and difficult to manage—one of the four deaths being almost entirely due to this—the ileum having some dozen small round ulcers in the midst of much acute catarrh.

Lastly, as to the clinical picture of the disease in the two towns. The difference here is most striking. The Manchester picture is the classical one—flushed cheeks, burning skin, restless tossing of the head whilst the body lies supine and still with outstretched arms, rapid and shallow breathing with no conscious dyspnœa, a brain on the borderland of busy delirium. But here the cheeks have a doughy pallor, the skin, though hot, can seldom be called burning, the decubitus is probably lateral though the child moves its body as a whole in a slow peevish manner, the breathing is more laboured than rapid, the cerebral excitement is much less, whilst abdominal pain, tenderness, and tympanites, along with a tendency to asthenia, are often present.

Such are some of the differences; the question naturally arises, Are these too great for the disease to be identical in the three towns? The best way to answer this will be to compare the points of likeness and unlikeness as to which are the more essential.

First, then, as to the *temperature*. We have seen the

differences. They are easily explainable. In Manchester all the lung mischief is done at once and ends at once; the march of the temperature is in accordance with this. Here we have a small outburst of lung mischief and a corresponding rise in temperature; two or three days later a fresh rise takes place, and on examination we find a fresh focus of inflammation has declared itself; finally, we have a long crisis or lysis, because these different foci, not having begun together, do not end at the same point of time. That this variation in the local lesion accounts for the difference of temperature is further exemplified by the fact that in those Manchester cases which were complicated by gastro-intestinal catarrh, the course of the temperature changes, becomes irregular, and approaches the Birmingham type, whilst the Birmingham temperature itself is much altered by abdominal disturbance. On the other hand, the points of likeness in the temperature in the three towns seem to me essential:—(1) The initial rise is probably in every case sudden, for  $\frac{9}{10}$  of the cases had a history of *sudden* onset; (2) the duration of the pyrexia is very nearly the same, crisis or lysis coming on the average in seven, eight and a half, and six days in Manchester, London, and Birmingham respectively; (3) the pyrexia once gone does not return, there is a subnormal reaction for a day or two (as one would expect, more marked in Manchester), and then the normal character is steadily resumed. These three points then—sudden inroad, equal duration, complete and lasting departure—are sufficient I think to determine the identity of the disease

as regards the temperature, notwithstanding its marked intercurrent variations.

Secondly, as to the *lung lesion*. In Birmingham it varies from a well-marked lobular pneumonia to a mere bronchial catarrh; it is essentially a lobar pneumonia in Manchester. This is not so easy to explain away as the varying temperature. Meigs and Pepper state that "the very same causes which produce lobar pneumonia in the robust give rise to bronchitis or perhaps to lobular pneumonia in children of feeble health." But this is evidently no explanation of difference due to locality; we could not for one moment admit that Birmingham children have feebler constitutions than their Manchester contemporaries; in fact this local difference of type tends to falsify Meigs and Pepper's statement. I am inclined to think it due to a more sthenic form of the disease being present in Manchester, as a result either of the *fons et origo mali*—whatever that may be—acting with greater intensity, or of the epidemic constitution, as Trousseau would say, of Manchester being of a higher pyrexial type. Virchow long ago admitted that a higher grade of disturbance may cause an exudation of fibrin into alveoli already partially filled with the cellular products of a catarrhal pneumonia; if then this higher grade of disturbance came at the *onset* we should have the double exudation of fibrin and of cells, the higher the disturbance the greater the proportion of fibrin, and *vice versâ*. Now an exudation of fibrin can occur more quickly than one of cells, hence, in the inflammation of highest grade the exudation is almost of pure fibrin, as

this has fully distended the alveolus before there has been any opportunity for more than a very few cells to be exuded. In favour of this view is the fact that in fibrinous pneumonia the alveoli are always widely distended. No inflamed alveolus has contents utterly devoid of cells nor quite destitute of fibrin ; the varying proportion of these two constituents is due merely to the local processes of inflammation, not to the nature of the disease.

But whatever the *cause* of this difference in lung lesion may be, it is admitted by most writers that the disease we call "acute pneumonia" can be accompanied by any form of lung inflammation, from a mere bronchial catarrh to a typical fibrinous consolidation. Rilliet and Barthez, after describing these various forms, go on to say : "All these different lesions of acute pneumonia are merely varieties of the same disease ; we have all the intermediate steps, and the one can transform itself into the other." Hensch of Berlin, in his masterly treatise on children's diseases, whilst agreeing in the main with Ziemssen's conclusion that there is a marked clinical contrast in the two types of inflammation, adds : "This is no doubt right for the majority of cases, but in no wise for all ; not every fibrinous pneumonia ends with a crisis, indeed a drawling course and transformation into a chronic form may occur ; whilst on the other hand I have sometimes seen pneumonias which gave a perfect picture of the catarrhal form, but yet had an unexpectedly sharp and healthy course, so that within five to eight days all was over." And again he says :



"Between the characteristic fibrinous and broncho-pneumonic types lies a middle form which has not yet clinically been sharply defined ; you cannot therefore in every case decide between the two types during life." This middle form of Hensch's, Rilliet and Barthez describe as "lobular hepatisation." Hensch quotes Steiner, Steffen, and Damaschino as having also shown that the products of a pneumonia, lobular in distribution, may be of a fibrinous nature. Steffen goes further, and wishes to employ the terms circumscribed and diffuse, instead of croupous and catarrhal.

There is nothing *sui generis* in fibrinous inflammation of a lung ; it is no more confined to the disease we call "acute pneumonia" than is the catarrhal form. It occurs as a complication of many other diseases. One of the most typical double lobar pneumonias I ever saw came on during an attack of acute rheumatism. I remember a man at St. Thomas's convalescing from typhoid who was suddenly seized with fatal pneumonia, and post mortem a typically fibrinous consolidation in the second stage, about the size of an orange, was found in each upper lobe. Hensch mentions a girl of twelve who died of typhoid, on whose temperature antipyretics had no effect, this remaining at 104° or over, and whose left lung was found post mortem to be almost completely solid. He also states that it occurs in other acute infectious fevers, especially measles,\* and that he

\* I lately took charge of such a case for Dr. Foster, where the onset of measles was complicated by acute laryngitis (for which tracheotomy was done) and double lobar pneumonia. *Vide* p. 133.



has not seldom found it in children who are suffering from tubercle and glandular caseation.

We may, then, take it for granted that difference in lung lesion is not of itself sufficient to disprove the identity of the disease; that "acute pneumonia" may show itself in the lung as an inflammation lobar or lobular in distribution, and fibrinous or cellular in quality.

These differences in lung lesion, then, weighty as they are, are not insurmountable, and there is this essential likeness—in all cases the morbid change is like a thunder-cloud which, though huge and threatening, leaves the sky as serene as it found it; indeed, as far as my experience goes, the chest of the Birmingham child loses all unhealthy signs at an earlier period than does that of the child of Manchester.

Thirdly, with regard to the *gastro-intestinal lesion*. This, though much more serious in Birmingham, is evidently of the same nature as that which occurs in Manchester. It is an acute catarrh of the alimentary tract, which sometimes goes on to ulceration just as the catarrh in the lung may lead to abscess and excavation. It affects the small gut chiefly, and has the usual stools of green slime and undigested food, with occasionally a trace of blood.

Fourthly, the greater *mortality* in Birmingham is only what would be expected from the differences which have been mentioned. An exudation of cells is much harder to absorb or get rid of in the sputum than one

of fibrin, and a lesion of a complicated tissue, such as the mucous membrane of the intestine, is far more difficult to repair than one of so simple a tissue as lung endothelium.

Lastly, in the *clinical picture* the likenesses are far more essential than the points of difference ; we have— (1) sudden onset occurring in all the Manchester, and in all but one of the Birmingham cases ; (2) this onset being accompanied by vomiting in twenty-six Manchester and twenty-seven Birmingham cases ; (3) the children are ill on an average about the same number of days ; (4) the recovery is rapid, though not equally so ; (5) and perhaps most importantly, this recovery is complete and permanent, there being no relapses.

These points of likeness are so much greater than those of unlikeness that they seem to me quite sufficient to establish the unity of the disease, and to determine that the Birmingham fever shall not be relegated to the uncertain limbo of broncho-pneumonia, the nature of the lung lesion notwithstanding. There is indeed no defined line of demarcation, for occasionally we get cases of true lobar pneumonia resembling the Manchester fever in every respect.

But if “acute pneumonia” remain one and the same disease though it wear such different guises, surely it cannot be merely a local malady. I left Manchester with all the dogmatic assurance of young ignorance that it was a lung inflammation pure and simple. But as house physician at the General Hospital I had

gradually and very reluctantly to admit that this thing could not be. How could I believe that a disease was but an ailment of the lung when I frequently saw all the physician's skill devoted to combating intestinal inflammation, or when my own examination told me that the nature of the lung lesion itself was of most varying character? I had only two alternatives—either the symptoms I saw belonged to different maladies or “acute pneumonia” was no local but a constitutional disease. From what I have said I need scarcely add that I gave in my adhesion to unity and constitutionalism.

After a few months more of observation I felt that I had not reached a logical resting-place. Not seldom I was bound to admit that the other acute catarrhs\* offered as conclusive evidence of a constitutional affection as did “acute pneumonia.” Still further did there sometimes seem to me a less wide gap of severance between such catarrhs and some examples of “acute pneumonia” than existed between different “acute pneumonias” themselves. Thus came to my mind the suggestion which I venture now to make to you, that “there is a constitutional pyrexial state which I propose to call catarrhal fever, which has respiratory, cutaneous, intestinal, meningeal, and renal varieties, just as there are pulmonary, anginous, nephritic, rheumatic, pyæmic, and cutaneous varieties of the constitutional pyrexial state called scarlet fever.” I

\* By catarrh I simply mean an inflammation of, leading to exudation upon, one of the lining membranes of the body.

make it not because I can go any length towards establishing its truth or untruth, but because I look to members of this Branch who have more experience than myself for counsel as to the best method of investigating its value.

I do not deny that there are purely local acute catarrhs ; one of the most powerful reasons for supposing that these local catarrhs are sometimes only outward and visible signs of a special febrile state being the very different course they take in those cases where we know they are the result of local irritation alone. For example, take meningitis, simple and tuberculous. The onset in the former is sudden, with high fever and all the signs and course of an acute disorder. It is a disease of short duration, death or health soon eventuating ; whereas in the tuberculous form the onset is insidious, the course much more prolonged and very variable, the symptoms corresponding in intensity with the amount of local disturbance produced by the tubercles.

Clinically I feel sure the recognition of such a general catarrhal fever would be most valuable. I remember, sadly enough, at any rate one instance where my narrow devotion to the lungs lost me my little patient, who died of enteritis. I was at that time unemancipated from the elementary notion that "acute pneumonia" was merely a local inflammation of the lung. Sir Thomas Watson long ago guarded us against this idea. Speaking of this affection he says : "You must not be guided by the mere *name* of a disease ; in

this particular instance you must not be guided even by the *thing*, *pneumonia*, itself. The constitutional symptoms must direct your treatment, while the local symptoms identify the disease."

We have seen that "acute pneumonia" is not specially connected with any one form of lung catarrh. But we may go much farther than this, and say that it may be complicated by every other acute catarrh from which the body suffers. (1) Pleurisy is nearly always present, not merely as a sequence from extension, but frequently appearing the earlier of the two; indeed, in children, even from the point of view of local manifestation alone, it is doubtful whether acute pleuro-pneumonia would not be the more correct expression. (2) The fauces I believe to be not seldom inflamed. Unfortunately this lesion is rarely looked for, except there be suspicion of some other febrile state; but even so, I find it mentioned in nine out of seventy-nine cases. If we bear in mind how often in the presence of so serious a malady a child may suffer a slight sore throat without complaint, we may fairly conclude that faucial catarrh is not at all infrequent; and Henoch tells us that slight throat pain, with hyperæmia of the pharynx and gums, is often observed at the commencement of the fever. (3) Catarrh of the alimentary tube has already been spoken of as a complication of supreme importance. The constancy of the initial symptom of vomiting—it is noted as occurring in sixty-five out of ninety-seven cases—shows how sensitive the stomach is to the shock of the onset. That



catarrh of the intestine may discover itself by constipation as well as by diarrhœa we know from enteric fever, where persistent constipation is almost as characteristic as looseness. Now in "acute pneumonia" this sluggishness of bowel is usually present, and it is especially apt to be the forerunner of a critical diarrhœa. Here this diarrhœa evidently indicates a resolution of an intestinal catarrh, just as redux crepitation and its accompanying expectoration point to a resolution of the catarrh in the lung. But the catarrh may chiefly affect the stomach, producing acute gastritis with persistent vomiting; or it may involve mainly the peritoneum. I admitted a girl of seventeen into the General Hospital in November 1884, who had all the symptoms of acute peritonitis. It was only on making the routine hospital examination of all the organs an hour or two later that I found the lower lobe of her left lung to be solid. Adolphe Baginsky, of Berlin, speaks of this form as gastric pneumonia. "Sometimes," he says, "severe gastric symptoms step into the foreground with violent vomiting and great diarrhœa of green offensive stools." Rilliet and Barthez state that we should look upon the diarrhœa which supervenes at an early period as a true complication: that in certain cases it is the indication of a colitis. Of such a nature was the lesion in the child I have previously mentioned as dying of enteritis. (4) Meningitis is of not infrequent occurrence. In February of this year a child was admitted to the General Hospital with lobular hepatitis of the left lung; brain symptoms were prominent, and though

the pneumonia speedily cleared up, the child remained seriously ill; a little later a squint arose, and death came in fourteen days; post mortem, well-marked simple meningitis was found. Dr. Ashby of Manchester told me a short time ago of a child to which he was called on the second day of its illness; it had had frequently recurring convulsions, and in one of these attacks he saw it; there were the physical signs of pneumonia in the left lower lobe. One hour later the child died convulsed. These convulsions were evidently not those due to the mere shock of onset; Dr. Ashby believed the case to be one of joint catarrh of lung and pia mater. Steiner has noticed otitis complicating a pneumonia, in which cerebral symptoms were well marked. Henoch says it is well known that cerebro-spinal meningitis can be united with pneumonia; but then, he adds, "the meningitic symptoms always remain the most prominent, and the pneumonia merely appears as a complication." This is scarcely a logical deduction of his, for whichever of the two be taken as the complicating or substantive lesion, the meningitis is pretty certain to occupy the foreground of the clinical picture. But there is a more numerous class of brain cases where the cerebral symptoms are persistent, but their thunder is subdued and muttering, with no sudden clap of onset; this is a well-recognised class, and is usually described under the heading cerebral pneumonia. There is, too, a series of intermediate cases—on the borderland. Two children were admitted to Pendlebury who had been suddenly seized with vomiting, rigor, headache, &c., a

few hours after a severe blow on the head ; in each, well-marked lobar pneumonia existed on admission ; both had unusually severe attacks, with high fever ; one suffered from persistent drowsiness, the other from active delirium. A third Pendlebury case, a boy of seven, was struck on the head a day or two antecedent to the onset ; he also had cerebral pneumonia, of which he died. Post mortem, all the organs were pale except the brain and lungs ; with regard to the former the note is ; "Brain, 45 ozs., veins and capillaries on surface gorged, substance firm and apparently gorged." Where then are we to draw the line between cerebral pneumonia and meningitis with pneumonia ? Surely it is more rational to look upon such cases as a joint catarrh of lung and brain. (5) Biliary catarrh probably all of us have seen along with "acute pneumonia." Trousseau designates this combination "bilious pneumonia," but Meigs and Pepper speak of "bilious *fever*" being frequently complicated with acute pneumonia. Which is right ? Is either ? Or should we not rather follow the golden mean and call them both mere local manifestations of the same diathesis ? (6) Acute nephritis is a rare concurrent of "acute pneumonia." Alexander Kees has a good article on this union in Jurgensen's treatise on croupous pneumonia. He has no doubt that they both arise from a common cause, and that one is no mere complication of the other. Klebs made the same assertion in 1875. By "acute nephritis" they of course understood something very different from the slight albuminuria which is almost constantly found in

the pyrexial state in children.\* (7) Hyperæmia of the skin—an erythematous rash—Henoch says can be often observed at the beginning. I only find it noted in three of my cases, in one of which the child was as “red as a lobster.” But these were not admitted till the fourth day, and the pneumonia rash is very transient. In the out-patient room, where I have also to supervise the casualties, I occasionally see it—usually as a blush on the front of the chest. Herpes labialis I need not mention, except to remind you that it is by no means always confined to the mucocutaneous junction, but is found on the centre of the cheeks and underneath the chin. (8) Lastly, there is the involvement of the larynx, trachea, bronchi, nasal mucous membrane, and conjunctivæ. These complications of “acute pneumonia” are so common and universally recognised that there is no necessity for me to do more than mention them.

“Acute pneumonia,” then, can be accompanied by numerous other local manifestations of catarrh, and the pneumonia need in no way be the most prominent lesion: indeed we have seen that catarrh of the cerebral meninges, gastro-intestinal tract, or larynx, usually takes the lead should it co-exist. The proportional importance of the lung lesion may dwindle down to a very small fraction. Can this fraction reach the vanish-

\* Dr. Payne, of Ilfracombe, when at the Children's Hospital in this town, told me of a case which he considered one of catarrhal fever, where post mortem the pelves of both kidneys were in a state of catarrhal inflammation, as well as the alimentary and respiratory tracts.

ing point? Can we have "acute pneumonia" without any inflammation of the lung? I do not believe such inflammation to be absolutely essential to the condition, though it is certain to be by far the most frequently present, as the cause of the catarrhal state—whatever that may be—is probably absorbed mostly by the respiratory tract.\*

But whether it be absolutely essential or no, surely it is not scientific nor broad-minded to call so multi-form a disease as "acute pneumonia" by the name of its most prominent lesion, nor is so doing best calculated to improve our knowledge of its nature.

All will admit that the lung lesion in "acute pneumonia" is no more characteristic of this state than is the joint lesion in rheumatic fever; but we should consider it a distinct retrograde step were we to style this latter "acute arthritis." The clinical picture of

\* This is at any rate no doubt the case in adults; but in children the alimentary mucous membrane must similarly act in a great number of instances; otherwise, how can we account for the frequency of acute gastro-intestinal catarrh in them? Speaking generally, then, we may say that in children the mucous membranes take up the catarrhal poison. But though the poison is first taken up by these, it does not always work its will on them; sometimes it is carried by the blood or lymph straight to the serous membranes without having set up any on-the-way inflammation in the mucous membrane which it has traversed. Just so does an individual convey the poison of scarlet fever from an infected person to a third person without himself participating in its imbibition. It is only in this way that I can explain the fact that acute catarrh of a serous membrane may occur unaccompanied by any detectable affection of a mucous tract, as, *e.g.*, may be the case in simple meningitis.



rheumatic fever is a far more complex one than is that of acute arthritis; it includes in addition pneumonia, pleurisy, pericarditis, endocarditis, and a peculiar sweat, as well as a remarkable subservience to salicin and a tendency to high temperature, &c. Now the clinical picture of "acute pneumonia" is more complex still; but the name through which we view it makes us short-sighted, so that we see its central features with even magnified distinctness, whilst the surrounding parts are blurred and ill defined.

On the other hand, can each of the other acute catarrhs be complicated in its turn by inflammation of the lung? As an answer I will simply quote the statement of Rilliet and Barthez: "There is not one of the diseases of childhood which cannot be complicated with pneumonia."

Again, with regard to the constitutional symptoms of these other acute catarrhs when they exist apart from any affection of the lung, there is a striking similarity between these and those of uncomplicated "acute pneumonia." Take acute tonsillitis, for instance. At Pendlebury I had the opportunity of observing the whole course of several cases of this fever which arose in the hospital. In its sudden onset, usually with vomiting and headache; in its duration; in its rapid rise of temperature and the high and persistent range of this temperature till its acute fall at the crisis; in the vanishing of all local pain as well as general malaise which followed at once upon this crisis, though the tonsils for some days longer remained swollen and

inflamed—in a state of resolution, in fact ; and in the speedy and complete restoration to health—in all these it strongly reminded me of the constitutional condition called “acute pneumonia.” At any rate, acute tonsillitis cannot be merely a local inflammation ; a sustained temperature of  $103^{\circ}$  to  $105^{\circ}$  cannot surely be caused by the inflammation of one or two small glands, of whose use in our economy we are as yet ignorant. Another point of significant interest is this : In Birmingham acute tonsillitis is less definite and sthenic than in Manchester ; it differs in type just as “acute pneumonia” differs in type in the two centres.\* Take, too, herpetic fever : its constitutional symptoms are almost identical with those of a light attack of acute tonsillitis or “acute pneumonia.” Yet the local manifestation is but the eruption of a few cutaneous vesicles. Are we to believe that these of themselves have a constitutional disturbing force equal to that produced by the solidification of half a lung ? Surely they can but correspond to the cutaneous rashes of the exanthems. Of the other acute catarrhs similar remarks may be made with equal cogency ; but as in these the local manifestation is greater, so the constitutional nature of the fever is less evident.

So far, then, we have come, and so far I trust will you agree with me, viz., that—

(1) Any acute local catarrh may arise in the course of acute pneumonia.

\* This I state as a personal opinion only, for I have collected no statistics concerning acute tonsillitis.

(2) Acute inflammation of the lung may arise in the course of any acute catarrh.

(3) All these acute catarrhs closely resemble each other in their constitutional symptoms.

(4) One seldom occurs alone : indeed three, four, or more are frequently present at the same time in the same individual. (Such frequent coincidence is, to say the least, hard to explain on the supposition that they are distinct diseases.)

But as yet the keystone of this bridge of argument is wanting. From a clinical, pathological, or therapeutic point of view there seems but little doubt of the unity of catarrh, but its etiology is a dull negation unless we believe that sudden fall of temperature is of itself a quite sufficient cause. That such difference is the usual *exciting* cause few I suppose will deny : few therefore will deny the unity of catarrh as regards its exciting cause. But if chill be but the exciting cause, what is the *fons et origo* which is brought into action through its agency? Is it single or multiple? Here, we must admit, our knowledge is of the scantiest. Yet what faint glimmerings come to us from the dimness of the unknown are in favour of this ultimate cause being one and indivisible. Pneumonococcus, though it is in my experience when present in vast numbers pathognomonic of lobar pneumonia, exists apart from this ; very few sputa of acute catarrhs fail to exhibit it, and we must suppose that it is only when it exists in excessive numbers that it can excite so acute a reaction as *fibrinous* inflammation.

Again, Eberth in a case of "acute pneumonia" found numberless small cocci in the sub-arachnoid fluid and in the tissue of the pia mater having the same appearance as those which he observed in the greyly-hepatised lung of the same individual, thus showing the power of these cocci to migrate throughout the body; if, then, they be the cause of the pneumonia, it is only rational to admit their power of producing meningitis. However, the etiology of the catarrhs is as yet untrodden ground, which should prove richly fertile to the persevering tillage of germvenators.\*

\* "Physicus, id est speculator *venatorque* naturæ."—*Cicero*.

### III. ON CATARRH

#### (B) ACUTE LARYNGITIS IN CHILDREN

IN a scientific classification of diseases no place should be found, I think, for the term Acute Laryngitis, as this is but a symptom of various maladies, chronic as well as acute. It is true that when the larynx becomes affected at the *onset* of an acute disease the result is often so striking that the other morbid symptoms are dwarfed, and we are apt, clinically, to speak of the whole affection as Acute Laryngitis, though this is, I believe, never even the sole *discoverable* lesion, but merely the one which intrudes itself most prominently upon our senses. But if, on the other hand, it arise late on in one of these same diseases—after other diagnostic symptoms have settled in our minds the true nature of the malady—then, though its severity may demand all our skill, and even put an end to life, we speak of it as a complication only. The child has, *e.g.*, suffered from measles *complicated* by acute laryngitis, just as this might have been complicated by pneumonia; whereas if the laryngitis have occurred with the first inroad of morbillar pyrexia, then has the patient been the victim of two substantive diseases—acute laryngitis and measles; and if further he should *die* before the outcrop of the morbillar rash,



the cause of his death will be written acute laryngitis only.

This surely is a most illogical state of affairs—one which must perpetuate hopeless confusion and sadly hinder us from being able “*rerum cognoscere causas*.” There can be but few cases of measles where laryngeal catarrh is entirely absent, but as a rule it contents itself with a very subordinate position, and we are content to ignore its existence altogether. The larynx is, however, of an irritable disposition, and sometimes resents this neglect by asserting itself in a most alarming fashion. Then we, taken aback, not having noted its customary more modest appearance, rush to the other extreme and are fain to acknowledge it as

“The very cause of Hamlet’s lunacy . . . .  
The head and source of all your son’s distemper ;”

instead of being merely an outward sign of the inward-working poison of morbilli.

None the less, however, is this its true position : it never reaches the dignity of a substantive disease any more than is the specific ulcer in the ileum the disease we call enteric fever, though a slit in its floor may end life almost as readily as closure of the slit in the larynx. Never, indeed, do I think a thoughtful observer can, with satisfaction to himself, write “acute laryngitis” alone upon any death certificate.

Now, I believe this to be no fanciful theoretical distinction, but one of extreme practical importance.

Allow me to adduce an example of its value :—A boy of six is admitted to the General Hospital suffering from urgent laryngeal dyspnœa ; tracheotomy is at once performed, and a large amount of undoubted membrane comes away through the wound ; the third day after admission the typical rash of measles appears ; two days later I see him for the first time, and find him in a state of high fever, cyanotic, a pulse of 190, and with both his lower lobes blocked with apparently true lobar pneumonia. My prognosis was, however, favourable, because my diagnosis was measles complicated by membranous laryngitis and pneumonia. If I had thought the child was the victim of the three diseases—diphtheria, measles, and that which we call acute pneumonia—I certainly should have prognosed otherwise ; for measles and double acute pneumonia would, I have little doubt, very speedily have put an end to a child who was struggling with the peculiarly fatal asthenia of diphtheria. Further, my prognosis was favourable, because I relied on the good effect of repeated doses of calomel and the continuous application of the cold-pack, remedies I could hardly venture to apply to one so liable to syncope as a convalescent from diphtheria. And again, when he took a happy turn, if I had believed in the diphtheria, my prognosis must still have been guarded because of the possible mischances of diphtheritic paralysis ; as it was, the croup was a thing of the past, and I had only the pneumonia of measles to consider.

The nature of acute laryngitis is evidently then of no

small practical importance, and this must be my apology for having so long and so egotistically detained this Society with its consideration.

With regard to morbid anatomy I shall say but little, for I have no new observations to record. With the acute submucous œdema which results from scald or sometimes occurs in the adult, I have nothing to do.

There are two forms of inflammation of the laryngeal mucous membrane. One is of a catarrhal nature and histologically is identical with catarrh of the nares, conjunctivæ, or bronchi; the other produces a felt-work of fibrin which encloses few or many cells in its meshes and adheres more or less firmly to the underlying mucous membrane which is itself infiltrated with a similar felt-work. This membranous form has many varieties; frequently its membrane is of a soft and creamy consistence and may easily be stripped off, or it is brownish, ragged, and closely adherent, leaving a superficially ulcerated surface when removed; the subjacent tissue may be but slightly infiltrated, or it may be extensively so, necrosis and sloughing resulting; various fungi are usually found amongst the cells and fibrin. But none of these varieties of membrane are of diagnostic importance, nor indeed does the presence or absence of membrane necessarily point out a particular disease as the cause of the laryngitis. There is no disease which may not produce membranous laryngitis, and diphtheria itself may run a purely catarrhal course. Such seems to me to be the gist of the many and confusing results obtained and

formulated by various recent observers and to it I give my hearty adherence.

This view cannot be more satisfactorily stated than in the Report of the Committee of the Royal Medical and Chirurgical Society on Membranous Croup and Diphtheria in 1879—"In fact," says this Committee, "it appears to us that the formation of false membrane in the larynx and trachea is merely a mode of reaction of the mucous membrane which may be set up by a variety of conditions." It is to the discovery of specific germs to which we must look for the power to causally differentiate the various acute inflammations of the larynx.

[From Article 6315 of the London Medical Record of Dec. 15, 1886, I extract the following :—In a discussion on diphtheria Guttman held that when the larynx is affected it is always secondary to the fauces, but that there is a true croup of the larynx which is primary. Henoch did not agree ; he held that an ordinary case of laryngitis, if severe enough, can end in membrane ; he had seen several such cases ; he had often seen croupous membrane on the fauces and larynx with diphtheritic at the bifurcation of the trachea, and hence concluded that they were different stages of the same process. In a paper read at a later meeting Virchow stated :—Affections of the mucous membranes may be divided into three divisions—catarrh, croup, and diphtheria. In catarrh there is a proliferation of the epithelium ; in croup there is a membrane formed by coagulation without any loss of substance, *nor can micro-organisms*

*usually be found.\** In diphtheria there is a necrotic process leading to the formation of a membrane, with a loss of substance beneath, while numerous micro-organisms can be found in the membrane and in the tissue beneath. The prognosis in a case depends on the presence or absence of ulceration. Diphtheria may be caused by swallowing acids or other irritants.† (*Verhandlungen der Berliner Medicinischen Gesellschaft*, 1884-85.)]

There is an unfortunate tendency at present to call all membranous exudations diphtheritic, which leads to a not infrequent diagnosis of diphtheria from the presence of membrane alone. But membranous exudation is apt to occur on all raw surfaces in low states of the constitution, *e.g.*, on unhealthy ulcers, or on stumps of low vitality. Mr. Nettleship, in his book on diseases of the eye, p. 335, says, "In the majority of cases of 'diphtheritic' and 'membranous' ophthalmia the disease is a local one, in which the disease takes on this special form. . . .

\* The italics are mine: "Nor can micro-organisms usually be found" = micro-organisms can sometimes be found. Now the amount in or frequency with which micro-organisms are observed is of no diagnostic value, as every advocate of the bacillar theory of phthisis must admit. It is merely an indication of the fruitfulness of the soil. The only point of importance is the *species* of the micro-organisms. Is it one? then are the diseases in which it is found one. Are they many? then is there diversity of disease. This I say, not of myself, but speaking as one who logically believes in the disease-producing power of bacteria.

† If these statements are correctly quoted—which I would gladly disbelieve—they seem to me unworthy of Virchow's greatness. The distinctions drawn are those of a mere morbid anatomist, not those of a pathologist. They help us nothing.



Many of the best marked membranous cases occur sporadically and without any history suggestive of infection from throat diphtheria or from any other exanthem." This is only an endorsement of Dr. Wade's statement in his classical article on "diphtheritis,"\* where he says, "I have never met with an instance of what has been described *under the name of 'diphtheritic conjunctivitis' in ordinary diphtheritis.*"

The diseases which may cause acute laryngitis are many : I shall mention some of the more important :—

(1) That pyrexial catarrh, which we are wont to recognise as due to chill, is perhaps the most frequent of all. It is here that we are especially liable to look upon the laryngitis as itself constituting the disease, but a brief consideration of the other symptoms attending on this state will, I think, suffice to disabuse our minds of this idea. The high pyrexia—often  $103^{\circ}$  or  $104^{\circ}$ —cannot surely be due to the purely local inflammation of a superficial square inch of mucous membrane, since no one has as yet suggested that the thermic centre has a laryngeal habitation. When I have sought it, I have never failed to find accompanying inflammation of the fauces ; nearly always do signs of large-tube bronchitis exist, and often there is pneumonia ; the coated tongue, almost constant constipation, and the vomiting which not seldom marks the onset, all point to involvement of the alimentary tract ; the trace of albumen which is generally at some time present speaks of renal conges-

\* *Midland Quarterly Journal of Medical Sciences*, April and July, 1858.

tion ; and lastly, the share of the skin in the disorder is not seldom shown by an evanescent erythema. All these symptoms then indicate that acute laryngitis, even when the result of chill, is but the most prominent feature of a constitutional affection. This variety of laryngitis is usually non-membranous ; indeed Sir Thomas Watson tells us that the presence of other catarrhal symptoms is strong evidence of the laryngitis partaking of their nature. But unfortunately the catarrhal state can produce a membranous as well as a non-membranous form ; it can cause an exudation of fibrin on the mucous membrane of the larynx as well as into the air-sacs of the lungs ; Virchow recognised this very clearly when he called acute lobar pneumonia croupous, to indicate the identity of its exudation with that of membranous croup.

(2) Diphtheria everybody believes has a special tendency to develop the membranous form of acute laryngitis, but probably by no means every laryngitis occurring in diphtheria is membranous ; and when the membranous form does arise it is not so deadly as that which results from the catarrhal state, for it does not possess in anything like the same degree the fatal power to extend throughout the bronchi. This is what we should expect ; the diphtheritic poison has its habitat in the fauces just as the enteric germs select the ileum, both we know can extend their influence either way, but then this influence has less effect than when exerted at their points of selection. Now the poison of chill appears to have no such selective powers ; it places the surfaces of

the body generally—external as well as internal—on the verge of exudation—that this exudation oftenest takes place through the lining of the respiratory tract is simply due to the fact that this is a very delicate membrane and one of those most exposed to the morbid action. It is but natural then that the mischief once started at a point should tend to spread in all directions. When the larynx is primarily affected the extension is mainly downwards along the trachea and bronchi owing to the delicacy of their lining epithelium, but it also pushes upwards, though the hardness of the pharyngeal mucous membrane renders this process tedious and limited. We may compare with this the wide-spread and inselective nature of the lesions in simple acute enteritis, a disease far more rapid and fatal than enteric fever, and one which is itself often the result of chill. The patches of membrane which thus not seldom appear on the pharynx in membranous laryngitis due to chill offer the greatest difficulty to diagnosis, and sometimes make it impossible to distinguish this condition from diphtheria.

(3) Measles frequently has acute laryngitis as one of its symptoms ; this may arise at the very onset, after the appearance of the rash, or during the desquamatory stage. West records 11 such cases, 9 of which were fatal ; 6 of these were examined post mortem and membrane was found on the larynx in 5, in the 6th the larynx looked granular and the epiglottis was ulcerated but no membrane was anywhere detected. [It is worthy of note, as showing the universality of the affection of

the respiratory tract, that in five out of the six post mortems double pneumonia was found.]

(4) The semi-membranous exudation of Scarlet Fever\* can, it is well known, extend to the larynx. Such cases when described are usually membranous in form, but then we must remember that mild laryngitis, not necessitating tracheotomy or other urgent treatment, would probably pass unnoticed in the presence of so formidable a malady as scarlet fever.

(5) Enteric Fever.—Only the other day I saw in the post-mortem room of the General Hospital a man who died in probably the third week of a severe attack of this fever. The larynx and its neighbourhood from the tip of the epiglottis to the bottom of the cricoid was almost completely covered by a rather thin, dry, dirty, light-brown membrane which broke off in small pieces and disclosed superficial ulceration of the subjacent membrane. The trachea and bronchi, so far as they were examined, were free, but a low form of pneumonia in the prune-juice stage existed in the lungs at both bases.

(6) Small-pox is mentioned by Sir Thomas Watson as a cause, though he does not state what form the inflammation takes.

(7) Facial Erysipelas Watson also regards as a cause.

(8) Injuries must receive a brief mention as causing membranous laryngitis. Hilton Fagge records seven such cases as resulting from scald.

\* For the morbid histology of this exudation read Dr. Crooke's report of his researches, *Birm. Med. Rev.*, Sept. 1886.

(9) Cœrtel seventeen times attempted to set up an artificial membranous laryngitis in dogs and rabbits by dropping a few minims of liq. ammoniæ on the trachea through an external wound; in every case he was successful, and the resulting membrane he was unable to distinguish from that of diphtheria.

(10) Lastly, acute laryngitis may develop in the course of a chronic disease, such as tubercle or syphilis; and often in these cases it has a tendency to take on the membranous form.

In dealing with acute laryngitis as it affects children only, there are three varieties where diagnosis of the true condition is extremely difficult and where, unfortunately, this is of very great practical importance. These are:—1st, That arising in diphtheria where the faucial lesion is but slightly marked and the laryngeal symptoms arise early; 2nd, Catarrhal laryngitis arising at the outset of an attack of catarrh or morbilli; 3rd, Membranous laryngitis arising in the same manner as the catarrhal form. In attempting their diagnosis I will, to avoid repetition, state the diagnostic points of positive value only.

In the 1st variety the history is one of gradual onset, usually at least three or four days. “*About* Monday he began to mope,” says the mother, “and went off his food.” Yesterday the difficulty of breathing was first noticed; this was not then severe nor did it seem greatly to trouble the child, but the difficulty has steadily increased; on inquiry you may find he has mixed with one or two others, adults or children, who have suffered



from sore throat. There is marked asthenia. You can hear but little air entering the bases of the lungs, yet is there no cyanosis nor strenuous effort to conquer the obstruction. The appearance of the face, and indeed the whole body, is one of grey pallor rather than lividity. The tongue is dry and brown whilst the bowels have tended more to looseness than constipation. The pulse is small, of very low tension, and very little disturbance alters its rate of beat. Pyrexia there is none, or but one degree or so. The urine has a thick cloud of albumen.\* The fauces whether membrane exist on them or not are but moderately inflamed, and there has been little or no history of painful swallowing.

2nd. The onset of laryngeal symptoms in catarrhal laryngitis is sudden and sthenic; the child has in no way ailed, or merely had a slight cold; he has not been ill enough to keep in bed or away from school. These are the cases to which one is hurriedly called at night, for often the child wakes with a fit of crowing.† Nearly

\* Dr. Wade, Sir Thomas Watson tells us, was the first to recognise the importance and constancy of albuminuria in diphtheria. But he did more than this; he showed (*loc. cit.*) that it was not simply the faint trace of albumen which, as far as my experience goes, may be detected in all the pyrexial states of childhood, but that it was, at any rate sometimes, a sign of true nephritis. "In some instances," he says, "we find what I should certainly have been disposed to anticipate in all, viz.: tube casts and renal epithelium. The tube casts may be of three forms, viz.: 'small waxy,' granular, and ordinary epithelial ones." It is this evidence of nephritis and the presence of a considerable amount of albumen which constitute the value of this sign as diagnostic of diphtheria.

† I am not now speaking of laryngismus stridulus, which is an affection I know but little of, never having had the chance of

always can you obtain from the mother, excited though she be, accurate information as to the hour or half-hour when dyspnœa first arose. This may not be the first or second paroxysm, and you may be told that no symptoms of ill health existed in between. There may have been previous attacks long since ; the parents or a brother or sister may have similarly suffered. The patient is evidently in the throes of an urgent sthenic disturbance ; the countenance is flushed, the eyes staring and bloodshot. Though the quantity of air entering the lung bases may seem to your ear far larger than was the case in the child with diphtheria, yet are most strenuous efforts made to increase this amount ; the lower ribs are violently drawn in, the nostrils dilated, the chin thrown out, and all the voluntary muscles brought to bear. Cyanosis is beginning, but yet pyrexia runs high and the pulse is full and bounding ; later, it is true, when cyanosis is advanced, the temperature drops and the pulse becomes slow and asphyxial. The fauces are acutely inflamed, the tonsils being usually much swollen, and deglutition is extremely painful. The tongue is clean and too red, or towards the base is a thick white fur, while the fungiform papillæ are unduly prominent near the tip. The bowels are confined. In the urine is but a trace of albumen, and this may only be discovered after two or three examinations.

3rd. This variety resembles the second in its sthenic nature and the suddenness of its onset. The history too watching a case, the attack always having passed its zenith by the time the child has reached the hospital.

may tell of a previous attack of catarrhal laryngitis, but this is unusual, although other members of the family may have so suffered. Whatever the previous history, the dyspnœa having once arisen is *permanent and increasing*, though, unfortunately for diagnosis, varyingly so ; on the other hand, its onset is more marked and its increase far more rapid than is the case in diphtheria. The conscious struggle for breath and life is here as painfully evident as in the catarrhal form, and the outward aspect of the sufferer much the same. No membrane, or but one or two small scraps can be seen on the fauces, and these are acutely inflamed and swollen as in the second variety, whilst swallowing is even still more painful. Pulse, urine, tongue, and bowels are in a similar condition.

Finally, a few words as to treatment. In this regard I shall refer solely to the three varieties just mentioned. Decision in these cases is of the utmost value. Our aim is to snatch life from immediate extinction ; that aim accomplished we have many resources by which to drag the child through the wreckage our somewhat heroic treatment has produced. Indeed, not much dragging will be needed ; children speedily recoup themselves for injuries received if the action of these be only of short duration.

Speaking generally, my routine method of proceeding is as follows :—At once on the child's admission I give an eighth of a grain of tartar emetic \* as a powder every

\* Powder, not wine, alcohol being contra-indicated and tending to prevent emesis.

ten minutes till free emesis occur ; if after five doses there be no emesis I double the doses at the same time that I double the interval between their administration ; if after three quarter-grain doses thus given no emesis occur I again double both dose and interval, but only twice in some thirty cases have I had to have recourse to doses of half a grain, and never have I had to give more than two of these. From the commencement, along with the tartar emetic, I give a grain of calomel every four hours. Should the case be a very acute one, and not unless, I place the child in a steam tent and if necessary keep *hot* sponges applied externally over the larynx. If these fail I ask the surgeon to perform tracheotomy. A castor-oil enema should be given immediately on admission. These are the measures I adopt for a fairly healthy child of two to four years ; they are those which were practised with such success by the preceding generation. In tartar emetic I have the greatest faith ; it is a most certain antiphlogistic\* and one of very rapid action ; I have never known it fail to produce emesis, a quality which greatly adds to its value in acute laryngitis, though in its emetic powers by no means resides its chief virtue, for great amelioration of symptoms often occurs before this is produced. Like most drugs of rapid action, its power is very transitory, and therefore calomel, whose changes are brought about slower but are more persistent, should

\* I have been called to task for the use of this old-fashioned word, but it seems to me to have quite as great a right to existence as the popular expression antipyretic.



be united with it, and its administration continued for at least sixty hours, or at any rate till all attempts at pyrexia have ceased.\*

In this relation I value the drug ipecacuanha at a very low price.† As an antiphlogistic I have never detected its power, and in acute laryngitis there is no time allowed for it to produce its special effect on the mucous membrane. It has emetic powers, but if emesis be the only end in view, sulphate of zinc has a simpler and more rapid action.

A steam tent and hot sponges constantly applied to the pomum Adami are remedies of the utmost value ; indeed, properly administered there can be but few cases of non-membranous laryngitis which they will not relieve ; but in private practice they are far less get-at-able than drugs, and there is often difficulty in instilling into the nurse's mind the difference between hot and *warm* sponges. They have one great drawback ; they merely alleviate the local evil, they do not strike at the general inflammatory condition which is the root of it all, as do the antimony and calomel. If once the urgent symptoms are combated by these I leave my patient devoid of anxiety, for relapses are rare and, when

\* I have never continued the calomel more than a week ; that is, have never given more than 40 grains. This I have never seen produce diarrhœa or stomatitis.

† I once gave a healthy child, who had just recovered from a slight attack of non-membranous laryngitis, *vin. ipecac.* ʒj, 2dis. horis for seven days. It was allowed to be up and to run about the ward as it pleased and was carefully watched by the Nurse and Resident Medical Officer as well as by myself, but we none of us could detect any action of the wine on the child.



they come, but subacute. But the steam and sponges do not touch this constitutional state, they merely keep it from manifesting itself locally in the larynx ; this too they keep in an enervating atmosphere so that it is more liable to catch cold again when removed from their gentle cossetting. It is always dangerous to take away a steam tent or the sponges at night, these should be removed on successive forenoons ; even so, however, it is occasionally as tedious a proceeding as the removal of a tracheotomy tube.

In a sthenic case I never therefore omit the antimony, whether there be membrane or no ; for I believe these prompt antiphlogistic measures go far to hinder the after-development of membrane in the bronchi and trachea, though I am aware some surgeons look with eye askance at these humble tributes of the physician, and long to bolster up the little fever-stricken child with iron and alcohol. This is because they believe that

All diphtheria is asthenia,

All membrane is diphtheria,

Therefore all membrane is asthenia,

which is a syllogism in Barbara, the soundest of all syllogistic forms ; but their minor premiss I deny *in toto*, for I maintain most earnestly that all membrane is *not* diphtheria.

Abstraction of blood would be useful when the administration of antimony is the less easy performance of the two, or in those extreme cases where even antimony's action cannot be tarried for ; but my

experience of it is practically *nil*, for I have tried it on but one case ; there it proved beneficial.

Lastly, as to tracheotomy.\*—I have never seen a case of non-membranous laryngitis which required this treatment, and it is hard for me to believe in its absolute necessity in this form.

Tracheotomy is no operation to be lightly undertaken ; in its results at any rate it belongs to the major rather than the minor performances of surgery. I have done it myself only twice, but have seen it performed a good many times by others, and it is especially true of so exciting a procedure as this, that lookers on see most of the game. My experience has lain too in three large hospitals where there is no lack of many-handed assistance, and where the operator has that greatest of all requisites—the skill resulting from repeated performance—in a far higher degree than most practitioners in private can possess. In those cases where there is only one medical man and no nurse I believe it to be an operation of the greatest danger. If you give no anæsthetic you have all the risks of a terribly restless child held in the unsteady arms of a loving novice. If you act both as anæsthetist and operator I need say no more to any one who has given chloroform on these occasions. However it is not tracheotomy itself but its results which I so greatly dread. When is the tube

\* Statistics certainly seem to show that intubation should be preferred to tracheotomy. It has been pretty extensively practised in America. Out of 180 cases collected by Northrup (*New York Medical Record*, Dec. 11, 1886) where this was performed for "laryngeal diphtheria" 55 recovered. The chief difficulty connected with it is that of obtaining a properly made tube.

to be removed and the hole closed? How long must the child run the increased risks which such an opening gives of pulmonary inflammation? When closure takes place granulations may still set up spasm or ruin the voice. In the case of non-membranous and membranous laryngitis due to chill\* you may have the whole bronchial tract in a state of acute inflammation and eager to produce a membranous exudation; the hole in the trachea lets in raw air, an irritating tube and teasing feathers upon this sensitive tract, and maybe gives the finishing touch to its membranous determination.

In non-diphtheritic membranous laryngitis then I put off tracheotomy as long as possible;† I have no fear of unduly depressing my patient; every case of this variety, which I have seen die after tracheotomy, has died of asphyxia from descent of the membrane along the tubes, not of asthenia. Here also is tracheotomy a most hopeless expedient; I have only known

\* I should express myself differently now: instead of "due to chill" I would say "due to the germ of catarrh."

† I do not underrate the tendency dyspnœa has to induce emphysema, nor the serious pulmonary weakness which results from emphysema arising in childhood. "As long as possible" then depends upon the severity of the obstruction; if this be great, and after two hours of energetic treatment considerable amelioration do not occur, I should have tracheotomy done. In one scale lie the present and after dangers of tracheotomy; in the other, the evils of emphysema. Correct balancing can only be obtained after much experience both in the post-mortem room and at the bedside. After all, emphysema is not so very frequent a result of this dyspnœa: even in fatal cases it only occurs in a small minority. Northrup (*loc. cit.*) found it in but 17 of the 87 sections made by him on children dead of "laryngeal diphtheria," and in several of these it existed to a very slight extent.

one out of ten cases recover, and she, it is significant to observe, was in extremis from long dyspnœa and collapse of lung before the operation was performed.\* Those that died usually got great relief from the operation and for a few hours appeared well, played and smiled, but in some twenty-four hours the deadly dyspnœa resulting from blocked bronchi began to show itself and the child died convulsed and comatose. The interim of healthfulness shows I think that the end was not brought about by asthenia.

In diphtheria it is otherwise. Here is asthenia enough and to spare. The membrane, too, has far less tendency to descend, hence the result is by no means so hopeless. It is not the progress of the local disease but the constitutional depression which one has to dread. Nothing can be more exhausting than the strugglings of dyspnœa. In diphtheria, then, I have tracheotomy done so soon as ever there be any serious laryngeal obstruction. In Birmingham I believe diphtheria to be extremely rare; during my residence at the General Hospital I did not come across a single example of the laryngeal variety. At St. Thomas's, where many of the tracheotomies were performed for this disease, the results were far more successful than here† simply because the membrane

\* She is now, eighteen months after operation, at the Jaffray Hospital, still wearing a tube (Feb. 1886). The tube has been removed but still occasional attacks of paroxysmal dyspnœa occur during sleep (Dec. 1886).

† I saw one day in the Victoria ward of St. Thomas's three children under two years of age who had undergone tracheotomy for diphtheria. They all recovered.

remained confined to a small area, and the asthenia could be successfully combated. Here the children die asphyxiated on the second or third day ; there they die collapsed in the second week.

Dr. H. W. G. MacKenzie, resident assistant physician\* at St. Thomas's, has most kindly sent me abstracts of a series of fatal cases occurring in that hospital after tracheotomy. As I think they well illustrate this difference in the two diseases I venture to, so far, reproduce them, classified (by me) under the two headings Diphtheria and Membranous Laryngitis.

## DIPHTHERIA.

A. L., *æt.*  $4\frac{1}{2}$ , death in seven days from asthenia with tracheitis and broncho-pneumonia.

H. M., *æt.* 4, death in 12 days from asthenia with broncho-pneumonia of right upper lobe.

A. S., *æt.*  $3\frac{3}{4}$ , death in eight days from paralysis of the soft palate and larynx with broncho-pneumonia.

S., *æt.* 33, death in six days from asthenia with pulmonary collapse and broncho-pneumonia.

## MEMBR. LARYNGITIS.

C. C. S., *æt.* 3, death in two days from dyspnœa. Membrane reached as far as the medium-sized bronchi.

M. S., *æt.* 4, death in two days from dyspnœa. Membrane extended to the smaller tubes.

J. A., *æt.*  $3\frac{1}{2}$ , death in 15 hours from dyspnœa. Membrane extending to the smallest bronchi.

H. B., *æt.* 12, death in 12 hours from dyspnœa. Membrane extending to the smallest tubes.

J. B., *æt.* 2, death in six hours from dyspnœa. Membrane extended along bronchi as far as traced.

\* Now assistant physician.



The remaining two I cannot definitely classify, though neither was probably diphtheria.

R. A. F., *æt.* 2, death in four days from very intense bronchitis and collapse of lung. No membrane found post mortem though the tube got blocked with it on the second day.

R. T., *æt.* 12, death in two days. Membrane extended to the bifurcation of the trachea.

In the October number of the *Birmingham Medical Review* for 1884 is a paper by Mr. Barling on the tracheotomies performed at the General Hospital during his residence as surgical officer. Ten times it was done for membranous laryngitis with one recovery; one death occurred on the twelfth and another on the fifteenth day after the operation, but no one of the other seven lived more than three days; these latter dying I suspect from extension of membrane to the bronchi, *not* from the asthenia of diphtheria.

I find in the same paper a rather startling list of eight tracheotomies for simple "laryngitis" with five deaths. This makes me all the more earnest in protesting against the operation in these cases. Two of the three recoveries developed whooping-cough, and had to remain in hospital twenty-two and twenty-one weeks respectively; the third was discharged on the forty-fourth day. Now, the longest stay which any of my fourteen non-membranous cases made was nine days, and he was treated with ipecacuanha; of those treated with antimony none remained more than seven, while the average length of stay was five, and in no instance was tracheotomy had

recourse to. As Mr. Barling's experience extended over two and a half years, whilst I was resident for nine months only, it may be argued that no case of such urgency as his came under my observation : this may be true, but my surgical colleague, Mr. Morrison, will bear me out when I say that on two or three occasions he strongly protested against my refusal to allow tracheotomy : such was the case also at Pendlebury, where my medical colleague, Mr. Chaffey, who had had several years' experience in children's hospitals, urgently combated my resolution to abstain from surgical interference. It is indeed the cordial agreement of my observations at Pendlebury, St. Thomas's, and the General Hospital which have rendered my statements to this Society to-night so dogmatic : for this, perhaps, unseemly dogmatism I ask their forgiveness.

### III. ON CATARRH

#### (C) ACUTE PRIMARY PERITONITIS

Acute peritonitis, as it has occurred in the medical wards of the General Hospital, may be divided into four classes.

- (1) Primary.
- (2) That due to morbid states of the blood.
- (3) That set up by extension from other organs.
- (4) Internal traumatism.

To-night I wish chiefly to speak of primary peritonitis. From a pathological point of view, it is by far the most interesting, as some lay doubt on its very existence. With your permission I will quote the opinions of one or two authorities in this particular; I shall then briefly relate one or two cases which are, I think, instances of this affection, and shall strive to show that the doubt and confusion has arisen from the idea that such peritonitis must be a purely local and isolated disease, and *not*, as I believe it to be, merely the local expression of a constitutional malady.

Roberts, 6th ed., p. 580, 1885, has six classes, the fifth he calls idiopathic. "Under this head are included those cases of peritonitis which cannot be traced to either class of causes above mentioned, but which are

attributed to cold, over-eating or drinking, and other injurious influences. Many doubt the existence of this variety, but exceptional cases come under observation which certainly seem fairly to belong to this class."

Aitken, 7th ed., vol. ii. p. 810, 1880.—"Its existence as a primary affection, like pleurisy, is generally ignored. But there is a class of cases which may be described as forms of idiopathic peritonitis in which effusion suddenly occurs into the peritoneum analogous to cases of latent pleurisy. These cases mostly arise from cold. They are generally sub-acute and often latent; in this respect bearing a great resemblance to the latent forms of pleurisy not uncommon in children and young persons."

Wardell, in Reynolds's System, vol. iii. p. 207, 1871.—"If peritonitis, like pneumonia and certain other diseases which formerly had always accorded to them an essentiality, is not to be deemed an essential complaint, it certainly, from its importance, demands a distinct place in a comprehensive work like that of 'The System of Medicine.'" "The causes of peritonitis are often traceable to wet and cold, damp feet, damp beds, chill winds, sudden alterations of temperature, as when, after being in a heated atmosphere, the body is rapidly cooled, or to excessive fatigue—in fact, to such general influences as are concerned in the production of inflammation in other viscera."

Alonzo Clark, in Pepper's System of Medicine, 1885, quotes Habershon's classical statement: "I cannot find a single case thoroughly detailed where the disease could

be correctly regarded as existing solely in the peritoneal serous membrane," and then adds: "It can, however, hardly be doubted that a much larger proportion of the cases are primary and idiopathic than either Louis or Habershon found reason to admit."

Sir Thomas Watson writes in much the same agnostic state of mind: "Much," he says, "of what I so lately said of acute inflammation of the pericardium may be said as pertinently of acute inflammation of the peritoneum. The membrane, when healthy, does not manifest any great or spontaneous readiness to take on inflammation. Peritonitis is often ascribed to some known exposure to cold, and especially to cold combined with moisture. But there is reason to believe that in most, if not in all, such cases some predisposing influence has been at work, some predisposition established, some previous unhealthy state of the membrane itself, or of the circulating blood."

Hilton Fagge strongly wishes to disbelieve in the existence of this form, but his sense of fairness baffles him. Here are his words:—"In some the cause is obvious; in others it may be utterly obscure, the patient having been supposed to be in perfect health until he is attacked by acute inflammation of the whole abdomen. Acute peritonitis is not in all cases traceable to an antecedent local disease. Sir Thomas Watson, in common with many of the older writers, gave exposure to cold as one of its causes; but general pathology lends no support to such an opinion." But though Hilton Fagge is so strongly convinced of the non-



existence of this affection, he is constrained to set down the opposite opinion of Dr. Fordyce Barker. To continue in his words :—"Dr. Fordyce Barker, indeed, speaks very confidently of having seen puerperal peritonitis caused by exposure to cold ; and *at least shows clearly* that in some cases no trace of suppuration or inflammation can be detected in the uterus or any of the neighbouring organs." Again : "In twenty years at Guy's there are recorded in the post-mortem reports only two cases of death from acute peritonitis in which the disease could not be attributed to any such causes as those of which I have been speaking." Here is an admission of two deaths, and primary peritonitis is by no means always fatal, as I shall presently point out. But Dr. Fagge goes further still, for, "In 1874, at a Wandsworth school several children were attacked with acute peritonitis *at the same time*. Dr. Anstie, who investigated this outbreak, came to the conclusion that it was caused by exposure to the influence of sewer gas. Dr. Shirley Murphy tells me he has lately met with a similar case. A woman, aged 36, died on her way to Homerton Fever Hospital, to which she was being mistakenly carried. At the autopsy, acute peritonitis was found to be the cause of death ; no local starting-point could anywhere be discovered ; the uterus, intestines and all other viscera were quite healthy. On inquiry it was elicited that the drain-pipe in her house had been blocked for two or three weeks, that in consequence the yard was covered with sewage, and some even lay on the passage leading to her room." These

were evidently cases of primary septic peritonitis where the inhaled poison passed harmlessly through lungs and pleuræ to spend its strength on the peritoneum alone.

Thus, with regard to the existence of acute primary peritonitis, there are some agnostics and some who remain faithful to the old creed, but none as yet who can call themselves positivists, believers in the new dogma of its non-existence. And, as my old master Bristowe lately remarked to me, the burden of proof lies with those who would deny that the peritoneum is liable to those general constitutional causes which set up inflammation in other serous membranes.

I will now detail a case of primary, uncomplicated, acute peritonitis which ended fatally.

CASE I.—Sarah Thomas, eleven years. Five days before admission she was suddenly seized with severe pain in the belly and with a desire to defecate. Straining at stool increased her pain and produced no motion. On the second day diarrhœa set in and lasted forty-eight hours. She vomited several times on the second and third days. On admission her temperature was  $103^{\circ}$  to  $104^{\circ}$ : the breathing was quick and thoracic: cheeks flushed: pulse small and hard: tongue had a thick yellow fur: the abdomen was hard and resistant, especially in the right lumbar and hypogastric regions. The temperature gradually fell to normal on the fourth day after admission, and she died on the sixth, that is the eleventh day of her illness.

*Autopsy.*—Brain and its membranes, heart and pericardium, kidneys, pleuræ and spleen all healthy. Lungs had slight collapse with hypostatic congestion of both lower lobes. Liver was pale and fatty. There was general purulent peritonitis: much yellow lymph with several ounces of pus. Intestines were healthy, having neither ulceration, rupture, nor strangu-

lation ; stomach also healthy. The ovaries and Fallopians were normal though matted with lymph. There was no vaginal discharge, nor any sign of caries or abscess about pelvis or abdomen.

This case may fairly be called, I think, one of uncomplicated primary acute peritonitis. That such are rare I need not say. In fact it is the only one I can find recorded in our post-mortem books\* during the last seven years.

But we have taken a very exclusive method of research. Judged by the same standard, primary pleurisy would similarly become one of the rarest diseases ; indeed during the same seven years I did not come across a *single* instance of uncomplicated pleurisy in the post-mortem records, unless a case of double empyema with great collapse of both lungs and considerable previous inflammation of these organs be allowed to stand as such.

Yet no one doubts the frequency of primary pleurisy. (I have not had time to inquire into the interesting question as to whether the older authors, before Laennec, deemed it as common as we do now.)

The reason is evident. Most cases potent enough to set up a *fatal* pleurisy would also have induced inflammation in other organs. A man has a slight chill and contracts simple pleurisy from which he easily recovers ; he has a severe one, and pneumonia, pericarditis, meningitis, enteritis, and peritonitis—one or more—are added to the pleurisy and then he dies.

I will relate one such fatal case :

\* *Id est*, of the General Hospital, Birmingham.

CASE 2.—Thomas Arms, 50, well nourished, no previous illness, six days before admission he was drenched in a thunder shower ; during the storm he says he was struck down by lightning and lay on the ground for ten minutes, he then got up shivering and walked home. His breath became very short and, during the night and next day, he was noisily delirious and remained very ill till admission. On admission he was stated to be moribund, examination impossible, but the pulse is noted as being small, very soft and compressible. At the section the state of things disclosed was as follows : Much yellow flaky lymph on both pleuræ with slight effusion of turbid yellow fluid into their cavities. Lower lobe of left lung solid, in the grey stage of pneumonia. Right lung congested and œdematous. Heart : 40 ozs. of clear yellow fluid in the pericardium with universal, thick, yellow lymph. Extensive general peritonitis : yellow soft lymph. Brain not examined. There was old peri-hepatitis and the kidneys were slightly granular.

It is just the same with peritonitis : a slight chill produces an uncomplicated attack and is generally recovered from ; a severer one adds the inflammation of other organs and the patient not seldom dies.

CASE 3.—Cicely Moffat is an illustration of this. She was a delicate child four years old and on admission had broncho-pneumonia, pericarditis, and peritonitis. At the section a pint of pus was found in the pericardium while a considerable amount of purulent serum was loculated in various positions in the peritoneal cavity. The pneumonia had to a great extent been recovered from.

CASE 4.—Clara Walters, six years, a child with bent tibiæ, but no previous illness. The day before admission she was seized with violent abdominal cramp, vomiting, and diarrhœa. On admission she was found to have double pleurisy, with



pneumonic catarrh and peritonitis. She died in fourteen days. At the section the left pleura contained some pus and flakes of lymph. The right pleura contained air with an ounce of pus at its base; the right lung being completely collapsed and carnefied. There was no evidence of pneumonia. The bronchial glands were large but not cheesy. The heart, pericardium, spleen, kidneys, uterus, and ovaries were all normal. General purulent peritonitis existed and the mesenteric glands were large but not caseous. There was some irritation of the intestinal mucous membrane but no ulceration.

These are not rare cases; we all recognise their existence. They by no means always die and the reports of the General Hospital contain records of not a few recoveries, two of which I will detail.

CASE 5.—A. F. B., a well-developed, healthy-looking girl of 16. She, however, had had an attack of acute rheumatism eight years previously, followed by recurrences in the three succeeding winters. The day before admission she had had several shivering fits with cough and frequent vomiting, and severe pain between the shoulders. At noon violent pains in the abdomen arose. Later in the day the breathing grew quick and shallow, the vomiting still continued, and the pain in both back and bowels increased. On admission I found her semi-delirious and constantly screaming, with both hands placed on the abdomen. Pressure upon this increased her cries. But though seriously ill she presented none of the extreme collapse indicative of internal traumatism. The pulse was quick but of fair size and rather hard; her face was flushed, and the thermometer registered  $103^{\circ}$ . Her breathing was almost entirely thoracic, and her abdomen acutely tender in all regions. So typically peritonitic did the symptoms appear that it was only some hours later, when making the routine examination,



that I found the lower lobe of her left lung was solid, and that signs of commencing pneumonia existed also at the right base. For treatment she had mercury and extract of opium, one grain of each, every four hours, with an ice-bag placed on her abdomen. The next day the pain was nearly gone though the temperature reached  $104^{\circ}$ . In ten days she was convalescent. There was no evidence of any cardiac or renal affection.

CASE 6.—J. Henry Towers, 14. Fifteen days before admission attacked with sudden pain, which was and has continued to be centred round the umbilicus. The next day the abdomen began to swell and has remained much distended ever since. The bowels were loose with two or three motions daily. He vomited once or twice but his appetite was good. He had some cough and expectoration. On admission he was poorly nourished. The tongue was clean and rather red. There was slight eructation with loose yellow stools, but no vomiting. The pain was still umbilical in situation and the abdomen was tympanitic and resistant with some œdema of its walls and its superficial veins were enlarged. There was also slight rubbing over the base of the left lung. For the first three weeks the temperature was variable, averaging about  $100^{\circ}$ , it then fell to  $99^{\circ}$  for some weeks longer. The diarrhœa also persisted and convalescence was protracted, but he eventually did well and left the hospital quite recovered.

The question is, how are we to classify these cases? In our reports the co-existing inflammations are all put down in the space set apart for diagnosis as if they were of equal value—none before or after other. And this seems to me the correct procedure; we may, clinically, diagnose the existence of one a day or so before the rest, but when the pathologist finds all the inflammations to be quite recent I think it wisest for us to consider them as all joint effects of one common cause. So considered

they would all be equally primary, and I have therefore classified such peritonitis as primary.

*A priori* reasoning is in favour of this view : it would be indeed strange if the peritoneum should be the only serous membrane in the body which was insusceptible to the inflammatory action of chill, and I know of no other which is not considered liable to its action. The peritoneum is identical in structure with other serous membranes and is exposed to the same evils as they : why should it not suffer then similar sicknesses ?

I therefore regard primary peritonitis as being usually one of the local expressions of the pathological condition brought about by chill ; just as this same cause produces pleurisy, meningitis or pericarditis.\* Rarely it is the *only* expression of this catarrhal state ; not seldom does it occur accompanied by a similar inflammation in some other organ.

Chill I believe to be its commonest cause, but just as we may have other forms of primary pleurisy, so may we have primary peritonitis arising from other causes.

For instance, those cases I have quoted from Hilton Fagge were no doubt instances of *septic* peritonitis.

Trousseau speaks of peritonitis developing spontaneously in enteric fever. Amongst the illustrative cases he quotes one from his friend Thirial, which is so peculiar that I venture to briefly reproduce it.

CASE 7.—A girl of 21, on the twentieth day of a mild attack of enteric fever and after convalescence had commenced,

\* *Id est*, chill is the predisposing cause.

was suddenly seized with symptoms of severe peritonitis and died in seventy-two hours. This onset of peritonitis followed upon a severe moral impression. Perforation was naturally diagnosed but none was found: there was nowhere even any intestinal ulceration; four or five darkened patches showed as many Peyer's glands in a state of resolution, but that was all. In no other organ was anything abnormal discovered. General purulent peritonitis existed, the mesentery being especially affected. This Trousseau appears to have considered a case of psychical peritonitis.

That it may arise in the course of the specific fevers is universally recognised: I have not observed such a case myself, but Dr. Line, of the Borough Hospital, has kindly sent me the following:

CASE 8.—A. M., 14, admitted with scarlatina anginosa. From the tenth day of illness green vomiting and diarrhoea persisted. There was no tympanites, but tenderness and retraction of the abdomen with marked facies abdominalis, delirium, and a small hard and irregular pulse. At the section general peritonitis with copious effusion of lymph was discovered.

All authorities allow that peritonitis is frequently secondary to renal disease. Hensch relates several such: one of these was in a boy eight years old, who had recently recovered from an affection of the joints, and who, during a hæmorrhagic nephritis was seized with an astounding succession of inflammations of serous membranes. First came an acute hydrocele of very severe character; then, after a uræmic attack, acute peritonitis occurred; and a few days later left

pleurisy, accompanied by much exudation, set in ; all these being proved at the autopsy.

Habershon, in a list of 262 sections in which peritonitis was found without any evidence of traumatism, states that sixty-two were due to Bright's disease. Now these cases are not supposed to be peritonitis from perinephritic extension, but to have arisen quite independently of local cause owing to the morbid state of the blood set up by the renal lesion. I cannot believe that the kidneys are thus able to act as the original and active focus for disseminating peritonitic poison.

That chronic Bright's disease impoverishes the blood and renders the individual far more liable to external malign influences is beyond question. But this is only another way of stating that peritonitis arising in cases of Bright's disease occurs in patients of enfeebled constitution. And this seems to me the correct form of expression, and the same holds good for any and every acute illness which seizes upon such subjects. Still it is not usual, nor would it be wise, to say that all sufferers from this malady die of it and not from the intercurrent affections which more immediately carry them off. Very few acute attacks arise in perfectly healthy adults, and the reason is not far to seek : such beings are *raræ aves* amongst us. But granular kidneys are no more the cause of peritonitis than is an enfeebled heart of pneumonia.

Such cases should also then come under the heading of primary peritonitis.

### III. ON CATARRH

#### (D) CATARRHUS

As the preceding papers indicate, in 1885 I held that there was a constitutional pyrexial state which, from its associations and for want of a better term, I named catarrhal fever, the cause of which was probably a germ and the exciting cause which permitted this germ to act was most commonly a chill; but that other depressants—*e.g.*, a blow, a nervous shock, or digestive upset—were also able to act as exciting causes. Clinically, its best example was acute pneumonia, but it received other names, *e.g.*, laryngitis, peritonitis, pericarditis, simple meningitis, when other organs bore the brunt of the attack. It was, in fact, a specific febrile disorder.

But in 1886 when writing to thank me for a copy of my paper on pneumonia, Dr. Wade made the following wisely-suggestive comment: "I have come of late years to look upon catarrh as one of the commonest of disorders, cropping up in an infinity of diseases and in many cases being the determining cause of death. Before you get your conception of 'catarrhal fever' too firmly fixed in your mind I would suggest to you to study the clinical history of *apyrexial* catarrhs, or those



which are nearly apyrexial. I do not say that such study will lead you to abandon your present views, but I think it will be useful to you as a practitioner and perhaps as a theorist." At first I felt a scientific resentment to this courteous criticism, but I soon perceived its force and was persuaded that in any theory of catarrh, those examples which did and those which did not produce a rise of the general body temperature must both be included. Recognising the unlimited scope of catarrh thus opened out and the multitudinous forms in which it appeared, I was at first non-plussed and lay by for clearer weather. I then, as now, looked upon catarrh as probably due to a specific germ, but at that time I considered an essential symptom of the action of germs was the raising of the general temperature of the body. I, in fact, believed in the *name* "specific febrile disorder" which by common consent was set apart for them and, moreover, believed that the adjective "febrile" was quite essential to that designation. But in the nine years which have followed much has been discovered regarding germs and, especially, the firm establishment of the unity of lupus, strumous glands and acute phthisis has done away for ever with the idea that germal action and pyrexia must needs go together; moreover, I have myself watched two cases of acute miliary tuberculosis of both lungs which ran their rapid course of six and eight weeks without any rise in the morning and evening temperatures, and I have both seen and read of apyrexial pneumonia, the most typically pyrexial of all the manifestations of

catarrh. A remarkable instance of this is recorded in the *Lancet* of July 11th, 1891, and certified to by two surgeons. A lad of 12 on board the P. & O. boat *Arcadia* had the whole of his right lower lobe and two-thirds of the left lower lobe solid, yet never was any rise of temperature noted, though this was taken thrice daily, while the lung signs cleared up and the boy convalesced in the ordinary manner.

So, too, most of us have seen cases of acute tonsillitis, acute laryngitis, acute bronchitis, where the carefully used thermometer has never registered 100°. How many of us have been almost disappointed when suffering from a heavy nasal catarrh which we confidently spoke of as a feverish cold, to find the temperature at its highest no more than 99°? As regards peritonitis and catarrhal nephritis, no one disputes that apyrexia is common throughout the course of these diseases.

In my own mind, therefore, when I think or speak of specific febrile disorders, I include all those diseases which have been shown to be, or probably are, due to specific germs whether pyrexial or not, the pyrexia being the clinical symptom of the active resentment of the body to the poisonous invasion. Sometimes this invasion is so intense and monsoon-like that the body is struck dumb and powerless and then collapse takes the place of pyrexia, with probably speedy death. On the other hand, the bacillary action may be so localised or feeble that a reaction sufficient to raise the general temperature of the body is uncalled for. It is evident that in all these three instances the disease is the same,

though the clinical pictures resulting may be widely different. Further, acute defensive processes would seem to be much more vividly set in action by injury to some organs than others. Lastly, we have to bear in mind the varying effect of the toxins upon a portion of the nervous system which probably controls temperature.

Indeed, were it not that pyrexia is such a striking symptom we should almost wonder that the term specific *fevers* ever arose. But when it was coined we knew but little of the class of diseases which it now designates, while the chief characteristic of those diseases which were universally included under it was far and away that of pyrexia. So much was this the case that Roberts in the fourth edition of his Handbook [1880] speaks of the phenomena of fever in this wise :

“First, they may follow and be the result of some local lesion in a tissue or organ, especially inflammation, when the fever is termed secondary.

“Secondly, they may constitute the chief and primary deviation from the normal state, not being due to any evident local cause, and, if any special organ or tissue becomes morbidly affected, this occurs secondarily. Idiopathic, essential, primary or specific are the terms applied to fever under these circumstances, or it is simply denominated a fever. It then originates from the presence of some morbid poison in the blood, either introduced from without, or developed within the body. The acute specific fevers and rheumatic fever afford examples of this class.” No doubt this language sounds

strangely ancient to your ears ; yet it is that of one of the two leading text-books of medicine which were in vogue in my student days. Now "*nous avons changé tout cela.*"

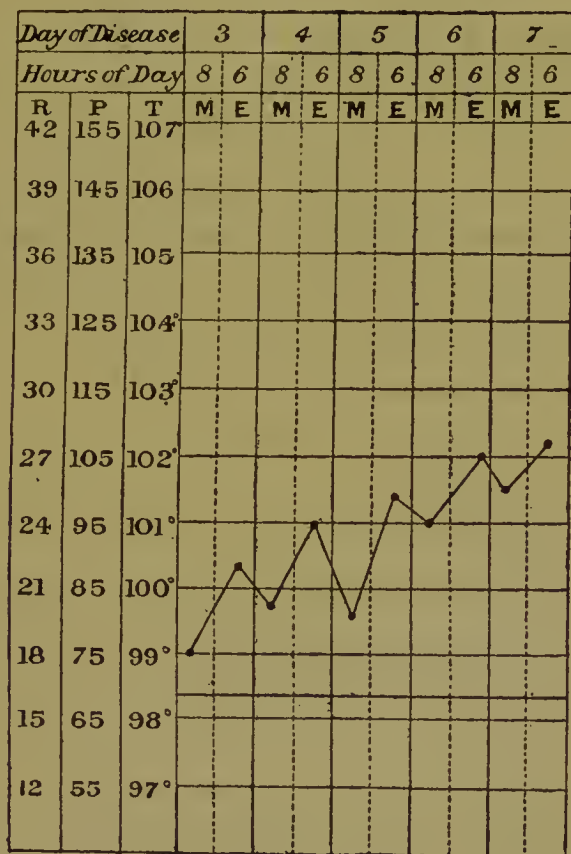
Metchnikoff has shown us that inflammation is the disturbance created by the defences which our bodies set up against the invading germs and their toxins : improved morbid anatomy has pointed out that, in fatal cases, the various local lesions which are the signs of these defences always indicate sufficient cause for the pyrexia ; above all, improved and multiplied clinical observation has discovered, in non-fatal as well as fatal cases, widely distributed and well-marked evidence of reactive inflammations, many of which, though formerly well known to be usually present, were described as complications or sequelæ, but which we now recognise as essential elements of the disease, indicating the foci where the battles between germ and tissue take place ; the varying intensity of such struggles determining the nature of the pyrexia, which along with the locality and other special characteristics of the disturbances, enable us to diagnose the nature of the invading germ, and thus name the disease it initiates.

Viewed in this light, pyrexia loses its mysterious importance and becomes merely the symptom indicating the total intensity of all the conflicts going on in the body at any one time. As this combined intensity tends to vary in a manner which is constant for each particular germ, the resulting pyrexia affords us a very great help in diagnosis. But unfortunately this tendency

is by no means an absolute one, and not seldom the temperature completely fails us as an aid to the elucidation of the condition of the patient.

What could be more typical of the first week of

FIG. 3.



enterica than this temperature curve? (Fig. 3.) Yet it is the record of the first week of a case of extremely acute tuberculosis which ended fatally three days later. Looking at these diseases from the modern standpoint, such pyrexial variation is by no means surprising: indeed we can only wonder that the course of the fever is



of so great diagnostic value as experience shows it to be.

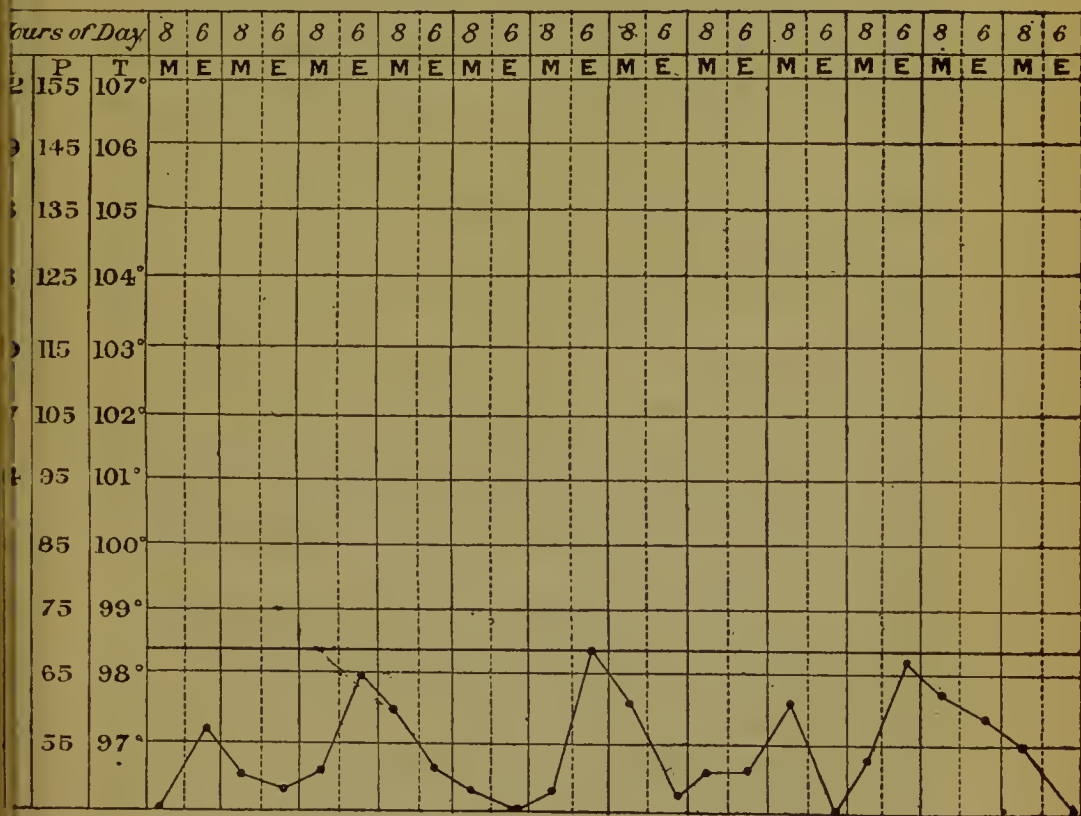
There has hitherto been much uncertainty as to what to and what not to include under the class acute specific febrile disorders. Roberts in the volume before mentioned consistently excludes tuberculosis and syphilis. Pye Smith, in his preface to Fagge's "Medicine," says: "I am not sure whether the author would have placed syphilis among the specific diseases or not. I should myself have done so and have put tubercle beside it." Bristowe includes syphilis but not tubercle. No doubt the name *febrile* diseases is responsible for much of this variability. Suppose for a moment we had a class of coughing diseases: is it not plain at once that probably no two of us would formulate an identical list of the conditions to be included under it? So must it ever be when we choose as our basis of classification a prominent symptom which is devoid of etiological significance.

Absence or presence of pyrexia, then, is no bar to the unity of catarrh.

But though there may be no pyrexia—that is, no general raising of the temperature above  $98.4^{\circ}$ —yet there is always an alteration of the temperature in this as in all forms of specific disease. If the constitution be feeble the shock of disease often makes the temperature take a subnormal range, but if defensive inflammation be in progress there will be periodic risings of the thermometer to  $98^{\circ}$  or  $99^{\circ}$ : the condition will be that known as subnormal hectic, a state which,

in my experience, never occurs without at least there being grave suspicion of organic disease. I once had hourly temperatures taken of themselves by several nurses at Brompton: the average diurnal variation was under  $2^{\circ}$ , but that of the night sister was  $4^{\circ}$ —from  $95.4^{\circ}$  to  $99.4^{\circ}$ ; she was warded for phthisis a month or two later. The accompanying tracing (Fig. 4) from

FIG. 4.



a case of pleuro-pneumonia is another example of this condition.

There thus opens out a very wide group of affections

which I would wish to unite under one designation. For the sake of clearness I will ask you to allow me to use a new term for this purpose, and the one I have chosen is *Catarrhus*.

Catarrhus, then, I look upon as a constitutional specific disorder, having many varieties of local expression, but I am inclined to think it need not always produce constitutional symptoms any more than does tubercle in the tuberculous affections of glands, or in lupus. It would seem that the toxins of its germ and of the bacillus of tubercle are often incapable of wide diffusion, or else that they cannot induce symptoms unless in a concentrated form, so that for the disease to become constitutional the germs must either exist in several scattered foci, or else have an unusually virulent nature. Some localised catarrhs, with frequent sub-acute recurrences, such as the pharyngeal and bronchial, may thus be cases of catarrhus, the small localised nests of germs undergoing repeated increases in their families whenever any depression in the tissue in which they are dwelling makes it a more suitable culture-medium for them. Similarly may we get frequent small outbursts of localised tuberculous mischief strictly limited to the apex of one lung.

But many—perhaps most—localised catarrhs are due to quite other causes, some quite independent of any germ and arising from chemical or mechanical irritation; some to the impaired nutrition of the affected membrane remaining after the ravages of a germ other than that of catarrhus, as measles, diphtheria, whooping-

cough ; some, again, resulting from the impaired nutrition caused by an acute attack of catarrhus itself, though none of its specific germs remain.

Thus the track of a tornado may be traced on the devastated land long after the atmosphere has resumed its wonted serenity, just as the damaged mitral remains to tell how rheumatic fever once held sway, and the pock-scarred skin witnesses to the disease whose terrors Jenner long since took away.

I would include under the specific disorder catarrhus the following affections : a variety of acute pneumonia, coryza, tonsillitis, quinsy, glossitis, sorethroat, laryngitis, croup, bronchitis, pneumonia, gastro-intestinal catarrh, meningitis, peritonitis, pleurisy, pericarditis, nephritis, hepatic congestion, vesical catarrh, and some skin affections, as herpes catarrhalis, some erythemas and urticarias.\*

I say a *variety* of these affections, for they can all occur in disorders other than the one under consideration, but then, clinically, their course is different. I never remember to have seen any one of these occurring alone

\* It has been objected to this category of affections, that it is so extensive that any disease which includes it all has denotation so largely writ that its connotation must be attenuated almost to a vanishing point. But it must be remembered : First, I do not include these affections as genera but only a *variety* of them all ; bronchitis, *e.g.*, is a name given to the genus of a local morbid process : this genus has many species : some of these species are complete affections ; but many are only the various bronchial manifestations of an equal number of constitutional diseases. Secondly, other specific disorders include just as large a number of local morbid expressions, only the *variety* is different to that which occurs in

in an attack of catarrhus ; and the combinations and permutations of their occurrences in individual attacks is endless. These combinations are a great argument in favour of the etiological unity of the things combined. Who amongst us doubts that the cutaneous and tonsillar disturbances of scarlet fever are etilogically identical ? Yet our certainty in this matter is due to their frequent combination in the disease and not because the specific poison has been found in these localities. It is true that often there is a special character in them when they occur in scarlet fever ; but, equally so, is there a special character in the respiratory and alimentary disturbances of catarrhus. No one will gainsay that, taking a large number of consecutive cases, the tonsillitis of catarrhus is as characteristic as that of scarlet fever. Further, as regards frequency of combination, I think the involvement of respiratory and alimentary systems in catarrhus is quite as frequent as that of cutaneous and alimentary in scarlet fever.

Moreover, this is not all : not only are these conditions frequently combined but sometimes one and

catarrhus. Take scarlet fever. Dr. Crooke, in those extremely valuable papers of his on the morbid anatomy of this disease (*Birm. Med. Rev.*, July, August, Sept. and Nov. 1886 ; June and July 1887) describes inflammatory affections of tongue, tonsils, throat, larynx, lungs, intestines, liver, spleen, kidneys, joints, as well as general acute adenitis ; and to these Dr. Line adds (*Birm. Med. Rev.*, March and May, 1887) coryza, otitis, cellulitis, peritonitis, stomatitis, herpes, and *the* rash ; to these again I would add pleurisy, meningitis and bronchitis. A list quite as large, you will admit, as the one I have above detailed. Yet no one, I believe, doubts the individuality of scarlet fever.



sometimes another element of this combination takes the chief position : and yet, and this is the important point, the general course of the disease is unchanged, though naturally modified in detail in accordance with the special lesion produced. Thus, Ashby, in Ashby and Wright's "Diseases of Children," describes the various varieties of pneumonia, his fourth being gastric pneumonia, which he thus defines : "In these cases gastric symptoms are most marked ; the attack may begin with vomiting, diarrhœa, coated tongue, fever and abdominal pain, and it is only after a day or two, when the classical signs of pneumonia appear, that a diagnosis of pneumonia is made." Surely this is no logical position ; "gastro-intestinal catarrh with some pneumonia" would be a name much more in accordance with the facts. But as yet we have not made up our minds to acknowledge a so-called idiopathic form of gastro-intestinal catarrh, so when we come upon it we restlessly hunt about till we find it in the company of some other affection, of whose right to the term idiopathic we have already agreed upon. Hence, though the pneumonia, even when it is discovered, occupies a very small portion of the disease-picture, yet we eagerly seize upon it as something we know, and insist upon calling the whole complaint by its name.

But to my mind the most convincing proof of the unity of all acute catarrhs lies in the definiteness of the *duration* and identity of the *course* of the disease, no matter what part of the body be the chief local sufferer. Take first the duration : this varies from three to seven

days, depending upon the severity of an attack : a mere coryza may last the seven, while a pneumonia may end in three ; and a peritonitis may be shorter-lived than a pleurisy. But whatever the severity or local condition, all varieties of catarrhus have as much right to the designation "One week " as typhoid has to its historical "Three weeks," and their existence is quite as determinate as that of scarlet fever or of influenza.

The unity of the course is even still more striking for, whatever the part affected, whether it be a coryza, the outcrop of a few vesicles, as in herpetic fever, or a severe pneumonia, always is the onset sudden—in a few *hours* the disorder is at its height ; there it remains for a few days with little variation : then, in as short a time as it took to rise, it vanishes, local traces remaining it is true, but the constitutional symptoms go completely, leaving only the lassitude of convalescence. Now this rapid vanishment, this crisis, does not strike us so keenly in simple coryza as in pneumonia ; but it is there, cut just as clear. Only the constitutional attack is slight in coryza and therefore its going does not strike us so vividly : moreover, the local repairs which have to be effected are on the surface and insistent, requiring a blowing of the nose for several days to come, and we do not consider our cold gone till this local annoyance has gone also. In pneumonia the local repairs take still longer : they are seldom finished in a fortnight from the crisis, but we neither feel nor see them, they do not intrude themselves upon us, so we conveniently forget their existence. Yet, let

the coryza be a severe and prostrating one, an influenza cold as it is sometimes termed—which by-the-by is a bad term, for influenza is an entirely distinct disease—then your crisis will be as marked as in pneumonia, for the constitutional symptoms are equally severe, and their equally sudden vanishment naturally produces an equal effect upon your senses, especially as in these cases the nasal discharge—*i.e.*, the local traces—often vanish at the same time.

You will say coryza more frequently relapses : I do not think so if it be treated with equal care. How often do we see, amongst the poor, evidence of relapsing pneumonia, against which the sufferer has vainly struggled, till at last he has been compelled to take to his bed. But pneumonia we usually put to bed at once, and if we did the same by coryza we should see or hear but little of relapses.

These very relapses even are an indication of the unity of catarrhus, for a relapsed coryza is not seldom a bronchial catarrh or a pneumonia itself. Is it not a well known laic saying “ his death was due to a neglected cold ” ? But a relapse or exacerbation does not change the specific nature of a disease. Hence in such cases at least the added pneumonia and bronchial catarrh can be but fresh symptoms of the original affection.

It has been often objected to the unity of catarrh of the upper air passages and acute pneumonia that the distribution of the two throughout the world is different ; but, on reading up the matter in Hirsch and Ziemssen, I cannot discover that this is borne out by the facts.

Unfortunately, nasal catarrh is too slight an affection to have its distribution well defined ; but bronchial catarrh is world wide though commonest at seasons when the atmosphere is damp and the temperature given to sudden depressions ; and therefore more common where this combination is frequent than where it is rare, no matter what the latitude may be. In our climate these seasons are late autumn and in spring ; in tropical climates they are those transition months which correspond to these.

Of pneumonia much the same can be said : it too is world wide : it too is commonest when damp and sudden depression of temperature are most frequent ; only, being, as I conceive it, a more severe attack of the disease, it is naturally more independent of external states and gives more marked evidence of its epidemic nature. Its season also is rather different, winter and spring, rather than spring and late autumn, being its favourite periods, though even this is open to doubt, and Hirsch suggests as one reason for its frequency in winter the confined atmosphere in which people live at this time of the year. In Switzerland, like catarrh generally, alpenstich (pneumonia) is commonest and severest at the melting of the snows, especially if the damp Föhn winds blow strongly then.

Further, we must remember that the statistics relating to bronchial catarrh refer vaguely as a rule to "catarrh and other pulmonic affections" as Daniell, a most accurate observer, styles it. Evidently here, then, we must include broncho-pneumonia, and if we agree to this, we cannot, as I have shown in my

paper on pneumonia, shut out the fibrinous variety of alveolitis.

Moreover, if there be some slight difference in the geographical distribution of the two, is this any bar to their unity? The varying incidence of tubercle in different latitudes is far greater. For instance, how rare is struma and other slow creeping and local forms in the tropics compared with England, and, on the other hand, with what much greater proportion does acute phthisis occur there than here. Indeed, what living thing, microbe or other, is unaffected by climate and will not modify its life history under the pressure of varying external circumstances?

Alimentary catarrh is also world wide: it, too, is to some extent increased by damp and sudden cold, though much less than catarrh of the respiratory organs; a more potent cause with it appears to be unhealthiness of ingested food. Of simple alimentary catarrh, it is indeed very difficult to get any definite statistics, it is so bound up and confused with other forms of dyspepsia, but when the catarrh occludes the bile duct then we get the well-marked sign of icterus which is very easy of identification. Unfortunately for my present purpose, this is an uncommon complication and though gastroduodenal catarrh is frequent enough, cases of catarrhal jaundice are comparatively rare and sporadic. Yet, when the character of the food would act specially and severely on the upper part of the tract, we should expect it to be fairly common in all those exposed to the same influence, as this portion would then



naturally be in all the most depressed and fall easiest prey to catarrh. Hence, we should expect epidemics of jaundice to be usually very localised and chiefly confined to those living on the same improper diet, and, moreover, that this diet should be not so much poisonous to the system at large as injurious to the stomach and duodenum. These *à priori* considerations receive striking confirmation from the researches of Hirsch. Of thirty-four epidemics, fifteen were confined to bodies of troops: three were localised to a ship-yard, a single house and a prison respectively: twelve were in a town or village; four only being more widely spread.

Moreover, the diets accused were either strikingly monotonous or indigestible in the ordinary laic sense; these two evil qualities would affect most the stomach and duodenum, for after them the food obtains the even consistency of chyme.

The whole matter seems to me clear enough. Some suddenly acting depressant is usually needed for the development of the poison of catarrhus in our bodies; by far the most potent depressant of the respiratory organs is the inhalation of cold and damp air, but this could have no special action on the alimentary tract; therefore, though "chill" has its etiological action on both respiratory and alimentary forms of catarrh, yet it will act far the strongest on the organs of respiration. Again, injurious matter passing down the œsophagus will exert its strongest action on the alimentary tract; hence we find unhealthy food and water are the most

potent exciters of alimentary catarrh and jaundice, that is, most strongly depress the digestive tract and so predispose it to succumb to the poison of catarrh.

Let us again take croup. Hirsch makes two forms : (1) Secondary or infective, occurring in typhus, measles, scarlet fever, small pox, whooping cough, and diphtheria ; and (2) true croup resulting "from harmful meteorological conditions." It is evidently the second variety which alone concerns us now, and regarding it he says it "generally goes with the acute catarrhal affections of the bronchial mucosa." He records twenty-five epidemics of it, but explains them as "merely a concurrence of numerous cases of croup at a particular place or district, and under the influence of the same meteorological conditions which so often bring about the more general prevalence" (or, as I should say, an epidemic) "of bronchial catarrh, slight or severe."

True croup and bronchial catarrh, from Hirsch's point of view, are thus etiologically one, but his single cause is chill, whereas I believe that chill is only the predisposant which allows the essential cause to act.

It is not to be understood from the foregoing that Hirsch concludes that "chill" and food are the *only* causes of respiratory and alimentary catarrhs. He gives most certain evidence that many other causes are sometimes at work—evil ventilation, evil drainage, exhaustion (*e.g.*, soldiers after a fatiguing march), racial predisposition (negroes very subject), and others.

Of the rarer catarrhs—cardiac, renal, peritonitic—the

very rarity with which they occupy the chief element of an attack of catarrhus prevents our learning anything of value concerning them from the ground of geography or meteorology; and when they arise as subsidiary phenomena to those conditions which usually take pre-eminence in the clinical picture, they naturally lose their individuality, the greater absorbing and including the less.

Thus geography and meteorology, far from speaking against, but add their testimony in favour of the unity of these acute catarrhs.

All these acute catarrhs, are probably mutually contagious. Pneumonia and coryza are generally recognised as being capable of reproducing themselves in a fresh subject, and it is also admitted by most of us that, given three men who are equally exposed to chill (*e.g.*), the chances are much against all three getting any one form of catarrh, but whilst one has pneumonia another one will get a head cold, and the third gastric catarrh. Further, it is agreed that though no doubt a cold may run through a house—that is, is infectious—yet this cold takes very different forms in the different individuals of the household. One has nasal catarrh, another chiefly shivering and general pains, a third a cold on the chest, a fourth an upset stomach. In this instance, at any rate, all these varieties of catarrh must be etiologically identical; and it is equally evident that this single cause cannot be “chill.”

On this point Dr. R. Percy Smith wrote me as follows in 1886: “With regard to the contagion of

ordinary catarrh, do you know the interesting fact that at St. Kilda, which is very much isolated from the rest of the world, they have an epidemic of what is called 'stranger's cold' whenever a ship visits the place, and never at any other time, although they are exposed to bad weather and storms?"

In the same letter Dr. Smith tells that at some schools which he at one time supervised there was an outbreak of various diseases, including facial erysipelas, acute pneumonia, and "thirty to forty cases of herpetic fever." The exciting cause of all these was, he thinks, defective drainage, and he goes on to make this suggestive comment. "I suppose all one can say is that the 'germs' of these different diseases happened to be knocking about searching for a proper nidus for development," intending me to conclude no doubt that the defective drainage was a depressing cause which afforded this "proper nidus."

At Pendlebury if a nurse in the scarlet fever ward developed a sore throat, though no other symptom of the fever, we did not hesitate to put it down to the scarlatinal poison, but if a nurse whilst nursing pneumonia patients develops a cold or sore throat, as she not seldom does, indeed with a frequency I think unaccounted for by the logic of chances apart from infection, we do not dream of calling it a pneumonia cold or pneumonia throat.

Further, many of us have known families attacked by scarlet fever where some individuals have had nothing but slight sore throats which would have passed un-

noticed at any other time, but which the dread sequela of nephritis has confirmed us in our previously hesitating diagnosis of scarlatinal throat. Similarly, for catarrh to exist in various forms in a household and for one member to suffer from acute pneumonia, I believe to be by no means rare, and have had personal experience of two of three instances of such occurrence, yet it never enters our heads to say that the acute pneumonia if coming first produced the other catarrhs, or if coming later was caused by one of them.

But I feel convinced that if once the contagiousness of catarrh were authoritatively recognised we should speedily have multitudes of examples of such contagion afforded us.

Another argument in favour of the infectious nature of catarrhus is the frequency with which all other causes fail. The action of chill is greatly over-rated : of pneumonia Ashby states that in Manchester it is almost equally prevalent at all periods of the year, whilst Fagge asserts that most authors who have tabulated their cases from this point of view fail to trace chill as a cause in any but a small proportion (59 in 603). And in speaking of our colds is it not an everyday saying, "I cannot make out how I caught it?" and so long as we determine to look only to chill as the cause of coryza, it is likely that this phrase will maintain its reputation.

Of the infectious nature of catarrh of the serous membranes it is harder to speak : it is very rare for catarrh to occur in them alone, and when it does, even if



infectious, it would have little chance to spread, if limited in its action to a closed sac. But there is the serous catarrh styled cerebro-spinal meningitis, and Sturges and Coupland suggestively point out the close relationship of this to acute pneumonia. Both conditions are infectious : are they one and the same ?

Infection from any disease of the alimentary canal does not often occur except through the stool, and there is good reason to believe that the summer diarrhœa of children is sometimes caused by such direct infection.

Hence, though we by no means have direct clinical proof, yet there is much to suggest that all these varieties of catarrh are mutually infectious.

Infection we can scarcely think of nowadays apart from a germ. If, then, all these various infections are to be included under one disease, the same germ must occur in them all.

Pneumonococcus has been found in all respiratory catarrhs, in meningitis, in pericarditis, and pleurisy, in peritonitis, in nephritis ; and cultivations of it have induced a condition resembling acute pneumonia when injected into an animal.

I can find no record of its having been searched for in the alimentary tract, but we cannot doubt that there it would be found as well. Indeed Flügge in his work on micro-organisms states that injections of cultivations of the "bacillus of pneumonia" into the veins sets up marked gastro-enteritis. It does this, it is true, in company with other germs, but I am not now arguing that pneumonococcus is the sole cause of gastro-intes-

tinal catarrh, but that one form of this affection is produced by its agency.

Its etiological value in peritonitis is much disputed, but Treves in his Lettsomian Lectures for 1894 on peritonitis makes it the cause of one of his five subclasses of this disease. He quotes Fraenkel as showing that in one out of twenty cases of peritonitis wherein he sought it, pneumonococcus was the only germ found. Also he states that Tavel and Lanz, Weichselbaum, Courtois, Suffit, Scavestre, Nélaton, Gaillard, and Monisse have reported cases of peritonitis where the pneumonococcus seems without doubt to have been the cause. In some of these the peritonitis was primary and the sole affection, in others it was secondary (*sic*) to ordinary pneumonia.

On the other hand, he tells us that Netter found pneumonococcus in the peritoneum in a hundred and fifty-one cases of fatal pneumonia, but that in only two of these was there peritonitis. I do not consider these results of Netter's much argument against the causative power of pneumonococcus. If catarrhus be a constitutional disease, then is the germ or its toxins scattered throughout the body and able to set up mischief in all parts. The peritoneum is but little obnoxious to its influence, else would peritonitis be as common as pneumonia.

Moreover, the germ of catarrhus must be even more omnipresent than that of tubercle: yet we are no more always being attacked with the disease than are we constantly the subjects of tubercle. The bacilli of

tubercle have been found in the expectoration of non-tubercular subjects: similarly may pneumococcus be found where no evidence of its evil power is discoverable.

Again, Treves remarks that irritants—*e.g.*, *tr. iodi* and *liq. ferri perchlor.*—produce peritonitis by so affecting the bowel that this allows bacteria to pass through its coats, which when healthy it will not do. It may be that the depressing irritation of chill or evil food may similarly allow the passage of pneumococcus.

There are several kinds of pneumonia bacilli: they are probably but varieties of the same species, for they are well-nigh identical morphologically, as well as in producing pneumonia experimentally; they agree also in most of their cultivations, the chief difference being in their growth in gelatine. Of these varieties those of Friedländer and Fraenkel are the most important. My remarks refer solely to Friedländer's, as I have discovered very few facts concerning the other.\*

Still the unity of these catarrhal affections and their specific constitutional nature by no means stands or falls with the capacity of the pneumococcus to produce them. The germ possessing this power may well be yet to find; and if on other grounds there be strong evidence of the existence of such a specific disease as

\* Drs. Washbourne and A. E. Wright stated at the Pathological Society on Feb. 5th, 1895, that Fraenkel's was the essential organism. Dr. Washbourne referred to his experiments in which after intra-peritoneal inoculations "peritonitis usually ensued."

catarrhus, this germal lacuna should only be an incentive to fresh germal research.

But whatever germ be the cause of catarrhus it must be, like the bacillus of tubercle, very largely dependent upon the suitability of the soil attacked. This suitability, as with tubercle, depends upon two things—inherited predisposition and an exciting cause, this latter being usually of the nature of a sudden depressant.

There are numerous individuals, otherwise robust, who are peculiarly liable to this disease: they “catch cold” on the least occasion. It is true that these colds are usually nasal, but that is merely because frequent bronchial, pulmonary, and intestinal attacks are not consistent with robust health, the individual suffering from them soon descending into the class of feeble lives, and thereupon not suffering so frequently, since such guard themselves more carefully against the exciting cause.

But though the inheritance of catarrhal susceptibility is undoubted, yet the germ of catarrh is pre-eminently, perhaps more than any other, dependent upon the aid of exciting causes. Far the commonest of these exciting causes is sudden lowering of temperature: so powerful, indeed, is this cause, and so understandable in its theory of producing the resulting inflammation, that it is hard to believe that it is not of itself all sufficient. Years ago I firmly held this creed, and I have given it up with great reluctance, but I have so relinquished it. More and more, as time goes on, we

are taught that in the production of acute disorders three causes are usually brought to bear—predisposing, exciting, and a germ. The exciting cause is the striker of the germal match to the laid fire of predisposition; and, indeed, as with the fire, so with the individual, this striking is the factor which is chiefly amenable to our control.

Still though the germ of catarrhus is thus usually so extremely dependent upon external conditions to work evil upon us, this is not always so. And herein lies one of the principal arguments in favour of its existence. Sometimes it does not lie like a dead match with potential vitality only, waiting the exciting cause to make it kinetic, but, unaided, it possesses the power to light up disease; to wit, when catarrhal affections become contagious, for a cold not seldom runs through a house, and pneumonia itself occasionally becomes epidemic. It is the existence of these attacks, arising apart from evidence of predisposition or exciting cause, which forms a chief bond of union between catarrhus and other acute specific disorders.

Moreover, the indubitably proved greatest frequency of catarrhus at periods of the year and in climates where sudden depressions of temperature and excessive moisture are chiefly concomitant, point to these conditions as being peculiarly favourable to the vitality of its germs, and to the supposition that the frequency of the disease at these times is in great measure due to unusual germal activity. The occurrence of enterica at special periods is similarly explained.



Catarrhus, then, I would define as an acute specific disorder of a week's duration. The incubation is short, seldom more than twenty-four hours and oftener only twelve: during it no special symptoms are discernible.

The onset is sudden, the height of the disease being reached in twelve to thirty-six hours: it is usually accompanied by chilliness or a distinct rigor, and vomiting is frequent. There are also headache and muscular pains.

The disease continues at its height for three to six days and, whatever form its local expression may take, the constitutional symptoms are very constant in duration and cease by crisis. If these should be prolonged, there is usually evidence of fresh local injury and the prolongation is of the nature of a recrudescence or relapse.

Pathologically it is essentially an exudation—serous, fibrinous, cellular, or membranous—from one or more of the lining membranes of the body. In severe cases some glandular organs become so seriously affected that acute interstitial inflammation, or even their suppuration, results. Some portion of the respiratory and alimentary tracts are always affected—the naso-pharynx being the commonest of all regions to be attacked. The tonsils and tongue rarely escape, quinsy and glossitis being the terms used when these regions are most severely involved. Next in frequency to suffer are the bronchi, and after them the pleuræ, alveoli of the lungs, and the stomach. Not seldom the intestines are injured

as well as the stomach, sometimes so severely as to cause ulceration, when the term enteritis is adopted. The pericardium and meninges of the brain are occasionally the chief foci of the disease: that covering the convexity of the cerebrum and, curiously, often only one-half of it, being the portion of the meninges which usually suffers.

The skin lesion is common and consists in a herpetic eruption, usually on the face, especially near the naso-labial junction, but occasionally on other parts of the body. Sometimes this forms the only striking lesion, and the condition is then styled herpetic fever. A transient erythema, limited to the chest and face, is by no means rare at the onset. The question of the nature of the involvement of the kidney tubules is a very complicated one: that they not seldom are one of the portions of the body to suffer is generally admitted, but whether they are often the chief focus of disease—that is, whether the condition called catarrhal nephritis is only one expression of catarrhus—is not so certain, though I believe this is sometimes the case. Peritonitis is without doubt occasionally but one of the local lesions of this disease, as is also a form of acute inflammation of the urinary bladder.

The crisis which puts an end to the constitutional symptoms occurs before any attempt at local repair has been attempted. It is evident that, as the parts to be repaired vary so much in character in different attacks, the time occupied by repair will also greatly vary: that is, the duration of convalescence is uncertain.

Moreover, it is equally evident that the signs and symptoms arising from the injury done to the parts of the body which have borne the brunt of the attack will vary with the locality and, when the organs affected are important ones, may so overshadow the general morbid state that the constitutional nature of the affection may fail to be diagnosed.

Pyrexia is usually present, and when present is characteristic. At the onset it rises suddenly to  $103^{\circ}$  or  $104^{\circ}$  in about twelve hours ; with irregular remittances it continues at much the same level till it suddenly drops below normal, when the constitutional symptoms of illness as suddenly cease. Thereafter is usually a day or two of subnormal temperature, followed by a few more of slight hectic (post-crisial hectic), whilst the brunt of repair to the damaged organ or organs is being accomplished. The remarkable constancy of this pyrexia, whether it be only the tonsils or a whole lung which is involved, points very strongly to the specific nature of the complaint.

Catarrhus is contagious, though this quality is never strongly marked. The incidence of its attacks nearly always depends upon inheritance and opportunity, as does that of tubercle ; but whereas inheritance plays the more important part with tubercle, with catarrhus opportunity is by far the most powerful. This opportunity is always of the nature of a sudden depressant, and is oftenest the sudden influence of cold acting in a damp atmosphere.

The essential cause is a micro-organism, in all

probability the pneumonococcus of Friedländer or Fraenkel, but its exact nature is not yet determined. It, like the bacillus of tubercle, is world wide in distribution and practically omnipresent where humanity is gathered together, but it only occasionally acquires sufficient virulence to successfully attack an individual in a condition of complete functional health.

It is not self-protective, in this particular being allied to tubercle and malaria, one attack increasing the predisposition of the sufferer to fall a victim to another.

Like other acute specific affections, restoration to apparently perfect health is the rule from a first acute attack; but as with them, so with catarrhus, the local damage may not be thoroughly repaired, and a debilitated pharynx may be left just as the ileum in enterica may remain permanently disabled.

You will perceive that I have for the most part confined myself to generalised statements, and have not attempted to clinch my argument by reference to illustrative cases or analysis of accumulated facts. Time is my apologist, not lack of these. As it is, I have almost unpardonably trespassed on your patience and, if from these I had sought assistance, this essay would have assumed the proportions of a lengthy monograph.

## IV. ON CLIMATE

### (A) THE DAVOS AND ENGADINE VALLEYS

THE Canton of Grisons is the most eastern portion of Switzerland : on the east and north it is bounded by the Austrian Tyrol, and on the south by Italy. Through the whole of this Canton runs, from south-west to north-east, the valley of the Engadine, which is seldom much over a mile in breadth and, often, as between Sûs and Zernetz, not half this width. It is divided into two portions, Upper and Lower ; the exact point of division is variously stated : Baedeker in his letterpress makes the Upper Engadine cease at Samaden, but in his map it extends to Zernetz. For our purpose to-night we shall include in the Upper Engadine only that most south-west district which lies between Samaden and the Maloja ; and it is to this Upper Engadine, so delineated, to which alone reference will be made.

The whole valley is some sixty miles in length, but the Upper Engadine measures only fifteen. This narrow tract of fifteen miles is one of great and striking beauty : its floor is chiefly occupied by a string of pale green lakes—those of St. Moritz, Campfer, Silvaplana, and Sils—the last and largest of which is four and a half miles in length. Linking these together flows the baby



Inn, rapidly growing by means of its many glacier tributaries, till it falls, a tumbling torrent, over the escarpment which so strangely separates St. Moritz from Celerina and Samaden. The edges of these lakes are clad with grass and bordered by charming walks beneath the shade of pines. On each side the valley the mountains rise steeply; on the north-west to end in the frozen peaks of the Piz Julier and Piz d'Err; while on the south-east lie massed the mighty glaciers of the Bernina range. But the charm of the Upper Engadine chiefly exists in the grand forms in which these mountainous ramparts are outlined: there is such striking diversity and boldness in their shapes that one can gaze long without perception of monotony.

Its places suitable for winter residence are—the Maloja Kursaal, Sils Maria, Campfer, St. Moritz, Samaden, and Pontresina.

The Maloja Kursaal is not very happy in its situation; it is nearly on the summit of the pass leading down into Italy; this is a very low one, and allows the south-west wind to rush up the Val Bregaglia with a good deal of its unpleasant moisture still in it. The Lake of Sils too lies very near the hotel, and is practically on the same level as this; hence one would think, as indeed I was told—I confess, at St. Moritz—the Kursaal is apt to share in the mists which hang over the lakes. Certainly on the night I spent there in October 1887, the whole place resembled a veritable cave of winds with all the winds let loose and a swirling snow-storm added. But Dr. Wise asserts that the Föhn (south-west wind) is rarely

perceived here. The Kursaal itself is a magnificent structure and far the most complete in its appointments of all the Hotels in the two valleys.

Sils Maria and Campfer are two charming hamlets on the south and north sides of the valley respectively ; they are both well protected from the wind, quiet and sunshiny ; as yet they are scarcely to be called winter residences, though Campfer is said to have an admirably conducted hotel.

Samaden has one good hotel which is kept open during the winter and is fitted up most completely as invalid winter quarters. Samaden is blessed with much sunshine, and is the Engadine capital, but the scenery will not compare with that of either of the other hamlets.

Pontresina is too bustling in summer, and in winter too exposed, to be suitable for any but very robust invalids ; for it is at the meeting of three valleys. For those who can stand such exposure, its large amount of sunshine would prove most exhilarating. It is scarcely as yet prepared to receive winter visitors.

St. Moritz Dorf is at present the place for invalids in winter, St. Moritz Bad is too exposed and, lying just at the upper end of the St. Moritzer See and on a level with it, is liable to the mists which cover the water. The village, however, is placed sufficiently high on the northern slope of the valley to be out of reach of these. The drainage runs into a reservoir near the Fall of the Inn ; here it settles, and the sediment is used for agricultural purposes, whilst the fluid portion flows into the river. In the village itself is nothing beautiful, merely

a straggling, stony, steep street, with uninteresting houses closely packed along its sides ; but the neighbourhood is delightfully romantic.

Some twenty miles to the north-west of that portion of the Lower Engadine which lies between Süs and Ponte, and running parallel to this, only sloping in the opposite direction, is the Davos Valley. It is separated from the Engadine by a mountain range of which the most prominent peaks are Scaletta and Kesch. The two are connected by the Fluëla Pass between Davos Dörfli and Süs, and by the Albula between Alveneu and Ponte. There is also a fine foot-walk from Davos Platz up the Dischma Thal and over the Scaletta Pass, which enters the Engadine by the lovely Salsanna Thal between Süs and Ponte.

The valley of Davos is only ten miles long ; it is narrower than the Engadine and far less interesting in natural scenery ; indeed, for a Swiss valley, it is distinctly plain and humdrum compared with its fascinating sisters. The Davoser See at its upper or north-east end is a small oval lake some two miles long, around which pine-trees cluster thickly and prettily, affording some charming points of view. This is the pleasantest part of the valley for, below, it consists of somewhat tame and monotonous grass slopes reaching up to the belt of pines, above which the eye sees only sky. When looking down the valley, south-westwards, however, some really fine Alpine scenery discloses itself, among which the Piz Michel, Piz d'Aela, and the Tinzenhorn—the Matterhorn of the Grisons—stand out conspicuously, well above the line of perpetual snow.

Dörfli clusters around the lower end of the lake ; it is the sunniest spot in the valley, but is more windy than the rest, being situated just where the Fluëla pass runs down. Dörfli is a quiet spot with few hotels, but several villas and cottages in which those whose pockets are shallow, or who love quiet, can find a very pleasant habitation. I came up from Landquaart with a lady who was returning to spend her fourth winter in a cottage at Dörfli ; she was loud in its praise, and her preference for it to the noise and bustle and smoke of "fashionable" Platz. This Platz with its much abused smoke and gaieties is only one mile further down. It lies at the foot of the northern slope of the valley, and is thus fully exposed to all the available sunshine. The inhabitants number some 5000, and the town consists mainly of houses dotted irregularly along the high road for the space of a mile ; these, if of any size, are either shops or hotels ; the shops are by no means poor, but their prices of course high. The drainage of Platz is excellent, both that inside the houses and the general system of the town. The unlimited supply of water enables the Davosers to do without ventholes. Through the drain of each house runs a ceaseless stream of water, and the main drain is kept constantly flushed by an impetuous mountain torrent. Thus any notable accumulation of gas is impossible. And as a matter of fact I failed to detect any unpleasantness in any of the closets of the Belvedere.

Davos Frauenkirch, three miles below Platz, is prettily situated at the entrance to the Sertig valley and just

below an old moraine which stretches half-way across the Davos valley at this point, but it is below the inlet of the Platz drainage into the Landwasser, and is moreover deficient in accommodation, the only Hotel being the Post, which is much like one of our old country posting houses.

Below Frauenkirch the valley contracts greatly, and has therefore too little sunshine to be suitable for invalids, though its beauty varies directly with its narrowness.

The principal hotels at Platz occupied by the English are the Kurhaus, Belvedere, Buol, and Angleterre. Of these the Belvedere and Angleterre are almost exclusively occupied by speakers of our language. The Belvedere has, perhaps, the best situation, and Herr Coester thoroughly understands making his guests comfortable. The Angleterre has a pleasant, genial English landlady and the furniture is of English pattern. These, with the cosy open fireplaces, make one almost feel in the country which, grumble how we may, we know in our inmost souls is, after all, far and away the best in the whole world. The Buol is lighted by electricity, which is of considerable advantage in the matter of ventilation. The Kurhaus is the only one which attempts any serious ventilation apart from door, window and stove. Indeed, ventilation is the serious drawback to Davos; and when we remember that from three or four o'clock in the afternoon to nine next morning must be spent indoors by the majority of the invalids, this drawback assumes very large proportions indeed. The Britannia dépendance of the Kurhaus admits fresh air from outside,



which is warmed by passing through a stand of steam pipes. These pipes are enclosed in a case made of layers of paper welded together to the thickness of a quarter of an inch, and the air enters the rooms through holes in the top of these cases. The amount of air entering can be regulated by the occupant. The paper case is so bad a conductor of heat that its external surface is scarcely warmed, however hot the pipes may be. But there are two grave objections to this system—first, it is used primarily to *heat* the chamber; when, therefore, this is sufficiently warmed, the air inlet is shut off and thus departs all attempt at ventilation. Second, there is no outlet for used-up air anywhere. When I naturally expressed blank surprise on discovering this I was naïvely told that you might open the window if you wished to get rid of the foul air. All these hotels chiefly depend upon upper fall-back windows; but the efficacy of these with stoves I found to be quite insufficient, my bedroom being stuffy on awaking in the morning, though my fall-back window was opened its widest. There are dotted about occasional tobins and wall-outlets, but these seem scattered haphazard, and some of them at any rate do not act. The Belvedere, at which I stayed, has four, fairly lofty, intercommunicating salons. I made a point of entering these just before the lights were put out—at 11 P.M.—and always found them disagreeably close. I suspect the atmosphere of the salons in the other hotels, whose rooms are less lofty, cannot, at any rate, be better in this respect.

The Kulm Hotel at St. Moritz is finely situated, looking down upon the lake and across it to the pleasant pinewood path to Pontresina, where the clear and cloudless Stützer See lies darkly hidden ; and then, farther south, the eye runs up the green pinewood slopes to the snow-clad summit of the Piz Rosatsch. But this is of summer that I speak, for in Mid-October, when I saw it, the whole was white, and as we left we saw Dr. and Mrs. Main starting the first toboggan of the season. The rooms of the Kulm are finer than any in Davos : they are extremely lofty and quite handsomely furnished. The saloon is an especially fine chamber with vaulted roof and elegant top lighting. In this room (as in the public rooms of the Kurhaus at Davos) there is inlet and outlet suitably situated. The ventilation of the bedrooms was no better than in the hotels of Davos, but as the Kulm rooms are larger and loftier the consequent disadvantages were correspondingly less. But I believe the energy of Herr Badrutt is going to change all this. At any rate in the new part, which he was kind enough thoroughly to explain to me, there is an elaborate and complete system of ventilation, in this wise :—The inlet of warm fresh air is practically on the same plan as that of the Britannia Dépendance of the Davos Platz Kurhaus, but here, in addition, is a properly provided outlet, which is situated near the floor at the opposite end of the room. The pressure of the steam in the pipes is only equal to half an atmosphere, hence the temperature never exceeds that of boiling water, and thus all excessive

drying of air is to a large extent avoided. As to this outlet being near the floor, it is so for the following reasons :—First, in winter the outside temperature is so low that were it near the ceiling there would simply be a rush of the pure warm air from inlet to outlet, and the floor of the room where the patient is would remain very imperfectly ventilated ; but with the outlet near the floor the pure heated air rushes up against the ceiling, and then, as it cools, slowly descends upon the occupant till, foul and cool, it slinks along the floor to the outlet, outside which, still comparatively warm and buoyant, it ascends above the house to be refrigerated and purified. Of the perfection of this method I have no experience, but both Herr Coester, of Davos Platz, as well as Herr Badrutt, assured me of its practical efficacy. One drawback to it is that the lowest stratum of air in the room is the coldest, and thus cold feet are likely to be perpetuated or else unpleasantly warmed brains. If the patient be confined to bed, it should of course act perfectly.

The great Kursaal on the Maloja has, I think, the best ventilation of all. Here the system is a central one : the air rushes over hot pipes in the basement and thence ascends through closed channels to the various chambers. The air enters each room close to the floor, the amount being regulated by the occupant. It is of a pleasant temperature, and hence *some* air is always required, and thus *continuous* ventilation obtained. The outlets are two in number in the wall opposite to that of the inlet : one is near the

ceiling, and is for the summer season chiefly, when simple unheated air enters by the inlet; the other, near the floor, is so placed on the same principle as that of the St. Moritz Kulm. Of the efficacy of the Maloja ventilation I made practical observation. On going to bed I shut window and door (there is no fireplace), opened the inlet and both outlets; in the morning the air was found to be perfectly sweet—so far as the sense of smell went—whilst the temperature had been kept pleasantly warm, allowing of very scanty bed covering. Yet I woke with none of the malaise from dry heated air to which I am usually easily susceptible.\*

Of the drainage system of the Maloja I know nothing, but the closet was quite sweet, and well appointed and heated. The other arrangements were excellent, and the public rooms very fine. Though we sat down but five to *table d'hôte*, I have nothing but praise to bestow upon the cuisine.

When discussing the value of mountain climates we have, amongst other things, to consider—

(a) **The Temperature.**—It is evident that the colder the inhaled air is the greater drying effect it must have upon the lungs. For the exhaled air has always the temperature of the body according to Steffen, or at any rate reaches  $85^{\circ}$  to  $95^{\circ}$ , as Hermann Weber thinks. Hence if air saturated with moisture at  $32^{\circ}$  be inhaled and heated to, say,  $85^{\circ}$ , it must exert more drying power than the inhalation of saturated air at  $62^{\circ}$ , which

\* Dr. Wise says that 3500 cubic feet of air an hour can be supplied to each person in the Kursaal when this is full—*i.e.*, has 500 inmates.

would be heated, say, to  $90^{\circ}$ , by the amount represented by  $53^{\circ}-28^{\circ}$ —*i.e.*,  $25^{\circ}$  F. For in each case the exhaled air is almost, if not quite, saturated.

(*b*) **Humidity.**—But apart from the low temperature, and therefore the small amount of *absolute* moisture in the air of mountain valleys—which in the case of Davos for the six winter months only averaged 3.2 mm. mercury and only 4.9 for the whole year—the *relative* humidity in these regions is very small; that for the winter six months of the year being  $81.5$ , and for the whole year  $78^{\circ}$ . But even this does not state the whole case, for, as one would expect, the air being so strongly heated by the reflected rays of the sun during the middle of the day, the relative humidity is at this time much diminished. For instance, in January 1887, the mean relative humidity of the month, taken at 7 A.M., 1 P.M., and 9 P.M., was  $88^{\circ}$ , but that at 1 P.M. only  $84^{\circ}$ . Again, in *June* 1887, the difference is far greater, for we find the numbers to be sixty-five and forty-six respectively. But as the average absolute humidity for June was 6.67 mm. mercury, whereas that for January was only 2.09 mm., it is probable that the drying power of the air was greater in January. This difference of relative humidity in different periods of the day is most important from the invalids' point of view. They are out only during actual sunshine—*i.e.*, during the time of least humidity. When indoors, what relative dampness there is in the air is speedily lessened by the increase of temperature the air undergoes on entering the heated apartment, so that the danger is of breathing not too damp but too



dry an atmosphere. It is this dryness of the air which partly causes epistaxis, as well as hæmorrhage from other superficial mucous membranes. These get dried up with a tendency to local stasis of blood in them, especially when the air is cold. However, this is only on first coming up, for (according to Dr. Huggard, the resident English physician at Davos) the mucous membranes soon acclimatise themselves to their new conditions.

(c) **Solar Radiation.**—The amount of this is really amazing. Dr. Frankland, so long ago as 1873, found at Davos on December 24th that a thermometer with blackened bulb, in the sunshine, in vacuo, at noon registered  $108.5^{\circ}$  F.; while on the same day at the same time at Greenwich it was only  $65.5^{\circ}$ , the temperature in the *shade* at Davos standing at only  $30^{\circ}$  F. This vast difference creates quite startling sensations in the residents in these valleys. Apropos of this Mr. de Beauchamp Strickland, editor of the *St. Moritz Post*, remarked to me concerning the Kulm Rink, round which the cleared snow was banked some eight feet high, that “sitting fronting the sun with your back in the shade, and facing this snow-wall, was like sitting over an English fire with a good draught behind you.” Such days of burning sunshine as these are the pink of the winter season, and in them, Dr. Main says, “we lunch on the ice with a snow-wall for a protection, under an umbrella to mitigate the heat of the sun, and with our water for lunch freezing in the shade cast by our bodies.” And again: “So hot is the weather that

many persons find it pleasanter to bask in the sunshine rather than indulge in the more active amusements of the place."

(*d*) **Barometric Pressure.**—This is some five inches less than at sea-level, which is equivalent to removing three tons from the total pressure on our bodies. Vapours diffuse more rapidly the lower the pressure, hence, other things being equal, evaporation will be much greater in high altitudes. Another reason this why the air of mountain valleys should be so dry.

(*e*) **Snow.**—This covers the ground during, practically, the whole of the winter season to the depth of three or four feet or more. It is therefore necessary to note its effect. It has these advantages according to Hermann Weber :—

1. The air-currents or winds resulting from the sun's heating the ground are almost entirely avoided.

2. There is no evaporation from the surface of the earth, for the snow has been cooled too far below freezing during the long night to be raised above it by the few hours of sunshine. The air is thus rendered much less moist and more diathermanous for the sun's rays, so that its illuminating, chemical, or warming power is much increased.

3. Emanations from the soil and ordinary dust are prevented from accumulating in (contaminating) the air.

4. Radiation from the earth is prevented and a limit set to the penetration of frost within it: the earth is kept warm, and hence quickly blossoms into summer

so soon as it and the sun become once again acquainted.

(f) **Sunshine.**—Listen to this extract\* from the meteorological report of Mr. and Mrs. MacMorland, visitors at the Hotel Belvedere at Davos Platz. Does it not make one long to cry with David and Mendelssohn, “O that I had the wings of a dove;” long yearningly for such bright calm and restfulness even as a miner longs to reach the light of day. It was January 1887—most of us can remember the month in England. At this valley of Davos they had twenty-three cloudless days, with only two on which any snow fell and this only three inches in depth, equivalent to one-fifth of an inch of rain. Sunshine in the world physical is like a merry heart in the mental world: it is Nature’s greatest tonic.

\* JANUARY 1887.—GENERAL DESCRIPTION.

1 Sunshine and clouds.	16 Cloudless.
2 Sunshine and clouds.	17 Cloudless.
3 Cloudless.	18 Cloudless save some cirrus.
4 Sunshine and clouds. Moderate breeze.	19 Almost constant sunshine.
5 Cloudy.	20 Sunshine and clouds A.M. Cloudless P.M.
6 Cloudless save some cirrus.	21 Cloudless.
7 Cloudless save some cirrus.	22 Cloudless.
8 Cloudless save some cumulus on the horizon P.M.	23 Cloudless.
9 Cloudy, snow P.M.	24 Cloudless.
10 Cloudy, with gleams of sunshine P.M.	25 Cloudless.
11 Cloudless.	26 Cloudless.
12 Cloudless.	27 Cloudless.
13 Cloudless.	28 Cloudless.
14 Cloudless.	29 Cloudless.
15 Cloudless.	30 Cloudless.
	31 Cloudless.
	Amount of water 0·21.

Light, says Hermann Weber, is inimical to the development of the bacteria and microscopic fungi which are associated with putrefaction and decay ; this preservative quality of light is most powerful in the direct solar ray, but can be shown to exist in ordinary diffused daylight. Tucker Wise has an interesting observation regarding the value of sunshine. He says many anæmic servant-girls come up to the mountains for their health's sake, and take service in the hotels. He has noted that those confined to the kitchens and shady rooms do not improve, whereas nurse-girls, who are out with the children every day, quickly do so.

(g) **Wind.**—This is one of the most important elements in climate, especially in high altitudes, where the air is piercingly cold. Here the slightest breeze is most keenly felt and changes a lazy basking in the sun to a desire for a quick walk and a warm overcoat. On going out after sundown I have particularly noted this—a very low temperature being borne with ease till a mild valley wind sprang up and made it almost unbearable. It was wind which made the cold of Vienna to me so unendurable. Indeed, the pleasantest day there was the coldest—4 below zero° ; whereas other days, when the temperature was as high as 10° and 20° F., a blustering wind pierced your very marrow and made outdoor exercise, as mere pleasure, impossible.

The winds in these valleys are of two kinds—local and general. The local or valley winds are morning and evening and correspond to the sea and land breezes of seaside health resorts. After sunrise the bottom and

lower slopes of the valley get heated sooner than the upper slopes and mountain tops, and they heat the lower strata of air, which thus rise and let down the colder upper strata and so produce the morning valley wind. In the evening the tops of the mountains and higher slopes radiate off their heat into space more rapidly than the more shut-in lower slopes and bottoms of the valley ; that is, they cool more rapidly, and hence cool the strata of air lying upon them. These thus descend, flowing down the slopes of mountains, and force up the not yet cooled lower strata, thus producing the evening valley winds. These valley winds blow with fair regularity and evenness of force and leave the middle of the day, when invalids wish to walk, practically unaffected by them. They are therefore easily reckoned with and are no drawback to the patient's existence. Moreover, they are necessary for the valley's ventilation : indeed, at Davos, it is doubtful whether a little more of them would not prove beneficial, as the air probably suffers at times from stagnation.

Of general winds the only one which exercises an evil influence, or is of strength sufficient to produce much influence, is the Föhn or S.W. wind. This is the deflected equatorial current and is more felt in N.E. Switzerland than anywhere else. Hermann Weber calls it a warm dry wind : "originally warm and moist," says he, "it loses most of its moisture on the western slopes of the Alps. Thus, in 1864, whilst the Föhn was blowing and the sky overcast, I noted the relative moisture was but 30 per cent. with a temperature of 82° F."



However, the Föhn is dreaded as a bringer of unsettled weather, of storms and clouds, of catarrh and dyspepsia ; besides it, even in winter, causes sometimes a slight melting of the snow. J. A. Symonds called it the "relaxing" south wind, but added that at Davos both this and the keen north are fairly excluded.

**Circulation.**—With regard to the effect of high altitudes on this, Øertel tells us that Waldenburg found the fulness of the radial pulse—*i.e.*, its sectional area—increased with diminishing pressure : so much did he find this to be the case that, whereas, under normal pressure, it took 41·7 cardiac contractions to completely arterialise the blood, under a very low pressure only 27 were required. Waldenburg also observed a considerable quickening of the pulse-rate.

The general arterial tension Øertel also finds to be considerably increased, and considers this due to, first, increase of cardiac force, or of relatively increased cardiac efficiency ; and, secondly, to the increase of the peripheral resistance owing to the greater fulness of the capillaries, on account of their being exposed to negative pressure. The absolute blood pressure is of course lowered, though not to so great an extent as the atmospheric.

This peripheral fulness of the cutaneous circulatory system naturally produces a rise of the peripheral temperature, *e.g.*, that of the closed palm was 2° F. above normal, whilst the central (*e.g.*, rectal) sank rather more than this.

But all these are laboratory and therefore temporary

results, and they do not tally with the observations on patients at Davos.

Theodore Williams quotes Ruedi—a physician of Davos—as stating that in 117 patients who had spent six months at Davos, the cardiac rate remained stationary in  $7\frac{1}{2}$  per cent., increased in 15 per cent. only, and actually decreased in 77 per cent.

But this again is delusory : patients are not healthy subjects ; the improvement in their general health might well so greatly lower the cardiac rate as to more than counterbalance the quickening effect of the high altitude.

At the same time, Ruedi's is the most important observation for us, as it is for patients we wish to use these valleys, and the result on them seems to be decidedly beneficial.

**Respiration.**—The effects of diminished barometric pressure on respiration are most confusing, and the results obtained by different observers, and even by the same observer, at different times, most conflicting. Especially do experiments made in the laboratory refuse to tally with those made on human beings residing at high altitudes (*e.g.*, those of Paul Bert, Cœrtel, &c., with those of Marcet, Jourdanet, &c.). Probably the following conclusion approaches to the truth. Other things being equal, a low pressure lessens the weight of oxygen in a given volume of air, and would therefore lessen the weight absorbed by the blood during a given inspiration, but the increased energy both of respiration and circulation at high altitudes more than compensates for this, so

that the interchange of oxygen and carbonic acid is actually increased—the profits made on a given capital at any moment are diminished, but the gain in a given time is greater, the turn-over being more rapid—a case of small profits, quick returns.

*What kind of Patients do well on the Alps ?*

(1) Those who want their chests expanded.—Hermann Weber, in fourteen young persons with narrow but not actually phthisical chests, found expansion varying from three-eighths to one inch after an Alpine residence of from three to twelve months. Theodore Williams found expansion in nearly the whole of twenty-two consumptives who had resided, on the average, six months (two to eighteen) in the Alps—chiefly at Davos. He supposes this is due (1) to hypertrophy—*i.e.*, a more complete development of certain portions of healthy tissue ; and (2) to emphysema of other portions, especially those which border on consolidations and cavities, these being unable to respond to the call of the deeper inspirations from lack of proper nutrition.

(2) Those who suffer from anæmia of those portions of the body which are exposed to the air, or who would be benefited by an excessive supply of blood to those portions.—That is, most cases of phthisis, especially those without fever, where the condition is one of stagnation rather than of inflammation. On the other hand, patients liable to acute inflammations of the superficies are, as a rule, unsuitable ; *e.g.*, those with a tendency to acute rheumatism, acute inflammations of the pharynx or larynx, or to acute bronchitis or

pneumonia. But cases of hectic not seldom do well ; abundant night-sweats, especially, often soon vanish. The air acts as a stimulant to the skin and remedies its relaxed condition. The great drying power of the air is also of advantage here.

(3) Those who require a dry atmosphere.—The exsiccating efficiency of the air is of use in chronic bronchial catarrh, in cases of secreting cavity, and in simple laryngeal catarrh. Caseous deposits should dry up. Hermann Weber has made eight post-mortem examinations on those who had temporarily or permanently been cured of phthisis by residence in elevated districts and he found chalky deposits in four of these. This certainly seems a large proportion ; but every pathologist will tell us that such masses are by no means rare in this country in lungs which show evidence of healed destructive processes, and, in the absence of extended observations, any dogmatism on this point is out of the question. By this I do not for one moment wish to deny the curative action of the drying power which Alpine air possesses, but merely to express a doubt as to whether chalky masses are found in greater proportion in cases cured by its aid than in cases of healed phthisis occurring in this country. Phthisis, when it heals, nearly always does so to some extent in this manner, wherever the healing takes place. That Alpine air is of great benefit in aiding this process hardly admits of doubt.

There are two classes of cases which resist all our efforts at exsiccation, and it is extremely unfortunate

that these are unsuited to an Alpine climate. I refer to chronic general bronchitis with secondary changes and cases of advanced bronchiectasis. All authorities, with one accord, assert the inadvisability of sending such patients to the mountains. In fact, whatever theories we may hold as to action of rarefied air and cold, practically there seems to be no doubt that the destruction of lung tissue must not be very great, that there must be a fair amount of breathing tissue left, and moreover, that the circulatory apparatus must be sound; this latter condition seems to be a *sine quâ non* for Alpine treatment. At the same time profuse bronchorrhœa without marked emphysema or fibrosis, slight bronchiectasis, or bronchiectasis *localised* to one lobe—*e.g.*, that resulting from an unresolved pneumonia—such cases as these do exceedingly well.

(4) Erethic cases do badly.—I mean by this those who are born with excitable irritable constitutions, whose temperature runs up on the slightest provocation, and whose mentality is proverbially mobile. Such constitutions as these run riot under the stimulation of high altitudes, and sadly wear out their possessors. But men made irritable by overwork or convalescing from severe illness recover fast, provided that previous to the overwork or illness their constitutions were fairly strong. Burney Yeo puts this well when he says, “I believe this kind of climate is especially useful to those who have been *strong*; but by some accident or other, such as overwork, or illness, or trouble, have become weak; to those who possess a latent power of reaction.



I do not think it so advantageous to the essentially weak person to whom 'twelve hours on a glacier' has been, is, and always will be an utter impossibility." A most striking instance of the truth of this was a young Cambridge man, who was the pride of the Belvedere when I was there in October 1887. He was expected two or three days after I arrived, and every one of the old residents always ended up their climatic adulations with the phrase, "Ah ! but you must wait till *he* comes." And I must say I was astounded. He filled the hotel with the noisy volume of his chest-notes, and towered above you a good-humoured giant, the very picture of athletic health. Four years ago he had been carried into the Belvedere and taken upstairs to—as some sympathetic young ladies told me—*die*; but seriously, he was supposed to be hopelessly gone in the advanced stage of phthisis, and an utterly unfit case for high altitudes. However, he rapidly picked up, and the next winter won the national toboggan race at Klösters, repeated this achievement in his third winter, and in his fourth ran second; and now, when I saw him, in his fifth, he appeared to have come up for pleasure as much as anything.

I think it is this condition of having some stamina, some backbone, which we must consider, far more than the actual amount of disease present. This granted, then there are few cases of phthisis, *e.g.*, which I would not advise to try Alpine treatment. We must always take *cum grano salis* the statements of the local physicians. They are doubly biassed. First, by the desire

to have a small death-rate ; an epidemic of mortality at an Alpine station would necessarily be fatal to its prosperity ; and, secondly, they have a praiseworthy shrinking from that quackery which lauds a certain drug as a universal panacea, and hence advise us, perhaps too strongly, in the opposite direction.

(5) Hermann Weber's statistics show that cases of hæmoptysis do well, and this I found to be the universal belief among the local physicians. Weber found that in 88 patients who spent in the aggregate 600 months in low-lying stations, 36 suffered from hæmoptysis, and that 62 attacks occurred during the 600 months. Whereas in 62 who spent 600 months in high altitudes hæmoptysis occurred in 11 only, and the number of attacks was 16. That is, the tendency to hæmoptysis in high altitudes was only one-third that in low stations. This is no doubt due to the lessened tendency to suppuration, septic states and rapid destruction of tissues, which an elevated climate produces.

*When to Go.*—As to the period of the year when patients should travel to these Alpine valleys I think there can be no question at all : it should be the middle of summer. This is the best time to travel and hence they are less likely to arrive with a chill. There will be less shock from change of climate : they will still retain in their systems a memory of our bleak spring so that the sharp nights of Alpine summer will not smite them too severely, and they will revel all the more keenly in the glory of its sunshine after the long winter of English discontent. Lastly, they will thus have become quite

acclimatised by the inset of winter and able to take full advantage of its benefits.

The great drawback is the much abused bugbear of snowfall in autumn. I purposely timed my visit so as to come in for this transition period, and I must say the danger and unpleasantness of this time is much overrated. You cannot rely upon absolutely settled weather anywhere about the equinox, but during the fortnight I spent in these two valleys I had only two snow-storms, no rain, no blustering winds, and as much sunshine as England would have given me in a month or six weeks. The air was keen, it is true, and after sunset nipped shrewdly, but it will be long before I forget the delight of smoking on the Belvedere balcony after breakfast; in fact the weather then was much like it was on the Riviera when I was there in January 1888. Just enough sharpness in the air to make one remember that lotus eating is not the chief end of our existence, and that exercise is good for the heart of man.

*How Long to Stay.*—Once up, stay there till you are well is the best advice. Far too much stress is laid upon the evils of snow-melting. Besides, these can be almost entirely avoided by judicious flitting from one Alpine station to another. For instance, the winter lasts longer in the upper Engadine than in the Davos valley. Let the denizens of St. Moritz or the Maloja leave their quarters when the snow begins to go and take the diligence to Davos Platz; or, better—for Platz is barely 1000 feet lower—fly to Wiesen, which, being high up on the northern slope of the magnificent gorge of the

Züge, quickly loses its snow when this begins to melt. The accommodation is excellent—equal to that of Davos; there are two hotels under one management and connected by a covered way. As to the proprietor, I can only say, with Dr. Main, “Herr Christian Palmy represents the Swiss hotel-keeper at his best.” But let me quote Dr. Main as to Wiesen in spring. He writes from there on February 1, 1888: “Change from St. Moritz seemed as one from winter to spring. The snow is already beginning to disappear fast, and we are able, for the first time for some months, to resume acquaintance with spongy turf. Seen in brilliant sunshine this gem of the Alps looks now at its very best. As a passage-place from the heights in spring Wiesen stands without a rival. Its climate is a delicious compromise, possessing the bracing air of higher regions and the more genial balminess of lower-lying lands. When the uplands begin to be shorn of snow, it will have its steep slopes carpeted with the richest of Alpine floras and its air laden in places with the odours of the lilies of the valley, which grow in wild luxuriance on many of its meadows.” Surely this is a mouth-watering picture of a spring abode, at least to us dwellers in Birmingham with the canvas of its climate so crowded with constant changes, and these, alas! so Plutonic that we cannot but be reminded of the various realms in Dante’s *Inferno*, and moodily groan out with Milton’s Satan, “And in the lowest deep, a lower deep still threatening to devour me opens wide.”

*Half-way Resting-places.*— But Wiesen is by no

means the only place of refuge ; the triple village of Tarasp, Schuls, and Vulpera, especially this last hamlet, is a most agreeable change from St. Moritz or Davos. It is situated in the Engadine, some dozen miles below Sûs, at an altitude of 4300 feet, and is unusually well protected from winds of all kinds. Thusis, at the foot of the Via Mala, and at an altitude of 2448 feet, is a good resting-place ; but it is too low for a prolonged stay and is more fitted for a halfway house, either going to or coming from the mountains, than as a mere change of abode.

Promontogno, 2687 feet, in the middle of the splendid Val Bregaglia, and halfway between the Maloja and Chiavenna, has a good hotel to receive patients, and is able, like Thusis, to act as a halfway house, or to receive fugitives from the snow-melting in the Upper Engadine—this is only one-and-a-half hours, eleven miles journey, from the Maloja. Just below it the road passes through a most picturesque gateway cut in the rock, which forms the upper limit of the chestnut, a fruit which forms the staple commodity of the pretty hamlet of Catasegna.

Seewis is delightfully perched above the valley of the Praetigau, about seven miles from Landquart, at an altitude of 3000 feet ; it has two good hotels, and forms an excellent halfway house and good spring resort.

*Davos and the Engadine compared.*—As to the rival virtues of these various spots they may be stated in a nutshell. The Maloja has the most sunshine and



perhaps the longest winter ; but it has these drawbacks : most wind, especially the Föhn, which rushes up the rocky rampart separating the Val Bregaglia from the Engadine—just over the ridge of which rampart stands the Maloja Kursaal ; and too great nearness to the lake of Sils with its cold damp mists.

St. Moritz has nearly as much sunshine as the Maloja, but this leaves it twenty to forty minutes earlier in the day ; about 3.0 instead of 3.30 ; this is a by no means despicable difference, as it curtails the walk after luncheon. But it is much better protected from the wind, and is well above any evil influence from the St. Moritzer See.

Davos Dörfli (5160 ft.) resembles St. Moritz in its sunshine and in the amount of wind, but its accommodation is far less luxurious, a perhaps not unmixed evil, as the evening entertainments at Platz and St. Moritz very likely do as much harm as good.

Platz (5115 ft.) has an hour's less sunshine than the Maloja, but is protected from the wind best of all, and, being 950 ft. lower than St. Moritz, is less stimulating and trying to the circulation. Socially, too, it easily bears off the palm. Some three hundred English reside in the hotels every winter, besides others living in lodgings and private villas. It no doubt will always prove the most generally suitable to the average invalid.

Finally, my advice would be, if you are strong enough, have sufficient backbone, go to the Engadine and by preference St. Moritz. If it grow too monotonous, drive over some fine day in January to Platz

and spend a month in Davos; then drive down with the first approach of snow-melting to Wiesen and stay there till June, when the Engadine again becomes delightful.

And thus with the same or similar variations remain up in these elevated regions for at least one year after all signs and symptoms of your disorder have vanished, and return to them again on the first appearance of any fresh outburst.

With reference to phthisis I would say, many cases no doubt get well at home, even amongst the poor. I remember patients at Brompton who had paid that hospital their first visit twenty and more years previously. But in these Alpine villages the *majority* seem to improve if they do not get well, and a far larger actual arrest takes place than in our own moist and low-lying climate.\*

\* Many changes have taken place in the last six years—including the railway to Davos. They may be summed up thus: These health resorts have become more popular and populous, and hence less suitable for invalids. Platz and St. Moritz have suffered chiefly in this respect: patients would do well, therefore, to consider the claims of (*e.g.*) Dörfli and Campfer, the desirability of which has improved absolutely as well as relatively (1895).

## IV. ON CLIMATE

### (B) AROSA : A HAMLET IN THE GRISONS, SWITZERLAND

IN the early part of October 1887, while staying at the Hotel Belvedere, Davos, I walked over the Strela Pass to Langwies, and, after a frugal lunch amidst its primitive prettiness, strolled up through the charming Aroserthal to Arosa. The pathway is a delightful one, running along the wooded western slope of the valley, with a constant view of the lofty and rugged cliffs of the eastern side which were sprinkled with snow and veined with miniature Staubbäche. The whole way is a long gentle ascent through woods and little grass-clad clearings, with their lonely chalets. The road itself was merely an ill-kept and muddy pony track, although it is not seldom traversed by sledges and ox-waggons. Indeed, a German lady whom I met at the Brunold told me she came up in a sledge, and was but waiting for suitable weather to return in the same fashion.

After walking for two-and-a-half hours from Langwies, on emerging from swampy footing and a thick belt of pine trees, one suddenly opens out the Seegrube or lower portion of Arosa, which forms half an amphitheatre around the charming little Untersee. On the

right, at the margin of the lake, stands the Pension Seehof, 5707 feet, well exposed on all sides to sunshine, but likely to be damp, lying as it does between the Unter- and Ober-seen. Opposite, some hundred feet above one, and a little back from the lake, is the Pension Rothorn, containing the post and telegraph office. To the left, on a level with the Rothorn, but finelier situated, facing north-west across the lakes and also north-east, is the best of the three pensions belonging to the Seegrube, the Waldhaus. All these were closed when I reached Arosa.

Proceeding westwards along the Aroserwasser between the Rothorn and Waldhaus, we reach in some half-mile the Brunold, 6000 feet, with its back against the Tschuggen Mountains and facing almost north. A little further on is the Kurhaus, the brightest and best kept of all, but also closed in winter time. It lies in an open spot, but has its principal rooms looking south-east against the hill-side.

The Brunold, where I stayed, is of the most unpretending nature, much more resembling a wayside inn than a place for prolonged residence. It is built almost entirely of wood, and is most primitive in arrangement; *e.g.*, the closet consists of a flat piece of pine perforated by a central round hole, from which goes down a perpendicular shaft formed by four pine timbers squarely fixed together. This shaft ended in a cesspool, the details of whose construction I could not investigate, as the snow lay somewhat thickly on the ground. I must confess, however, that I discovered

no disagreeable odour, although it seemed to bear unaided the whole burden of the Brunold's excrement; and even then, in October, we numbered some dozen souls. But the accommodation, though plain and thoroughly German, was excellent of its kind, and our hosts were full of genial courtesy. I met three German ladies who were intending to winter there, with the phthisical husband of one of them. In 1886-1887 two chest invalids had similarly stayed.

A little farther off, and a good deal higher, was a dwelling-house where a resident had constantly lived some two years. He had built the house after having benefited during the previous winter by residing at the Brunold.

The only meteorological statistics I know of concerning Arosa are found in the "Correspondenz-Blatt für Schweizer Aertze Jahrgeschichte," xvi. 1886, by Fritz Egger, a Basel student who, with his brother and a friend, all three suffering from phthisis, and an English clergyman who had over-worked himself, spent the winter of 1885-6 at the Pension Brunold. In brief they are these:

**Temperature.**—This was taken at 7, 1, and 9 o'clock.

	Nov.		Dec.		Jan.		Feb.
Monthly average recorded .	33°	...	24°	...	20°	...	20° F.
Monthly minimum recorded	17	...	4	...	1·5	...	0·5 „
Monthly maximum recorded	45·5	...	46·5	...	37	...	39·5 „

Observations taken hourly for one week showed that the daily variations were, naturally, much less than those recorded above.



At Davos Platz similar observations during the same winter gave the following results :

	Nov.	Dec.	Jan.	Feb.
Monthly average .	34·7°	24·8°	21°	23° F.
Minimum average .	13·5	3	5	6·5 „
Maximum average .	52	50·5	45	44·5 „

—from which it would seem that while Arosa is somewhat colder than Davos, yet its variations in temperature are much less, and would more than compensate, from an invalid's point of view, for its extra coldness.

**Cloud.**—With regard to the amount of cloud, it again seems to possess some advantage over Davos, as the following tables show. The cloudiness is measured in numbers from 0 to 10 ; anything below 2 is reckoned a clear day ; anything above 8 a cloudy one.

	Nov.	Dec.	Jan.	Feb.
Arosa, 1885-1886 :				
Clear days, 0-2	. 12	... 18	... 9	... 16
Cloudy days, 8-10	. 7	... 6	... 10	... 4
Amount = 0-5	. 18	... 21	... 15	... 21
Amount = 5-10	. 12	... 10	... 16	... 7
Davos, 1885-6 :				
Clear days, 0-2	. 10	... 16	... 10	... 17
Cloudy days, 8-10	. 4	... 7	... 10	... 5
Amount = 0-5	. 21	... 20	... 17	... 20
Amount = 5-10	. 9	... 11	... 14	... 8

**Snowfall.**—During 1885-6 the days of snowfall were fewer than at Davos, thus :

	Nov.	Dec.	Jan.	Feb.
Arosa, 1885-6 :				
Days with snow .	. 6	... 7	... 6	... 4
Davos, 1885-6 :				
Days with snow .	. 7	... 11	... 12	... 8

**Wind.**—The upper wind occurs very seldom ; it either comes as a severe *Föhn*, or as an east wind, *Orkan*.

	Nov.		Dec.		Jan.		Feb.		
Arosa, 1885-6 :									
Föhn days .	.	.	1	...	1	...	0	...	0
Orkan days	.	.	0	...	1	...	2	...	0
Davos, 1885-6 :									
Föhn days .	.	.	3	...	1	...	3	..	2
North wind days	.	.	0	...	1	...	0	...	2

In these two tables the north and east winds may be said to balance each other, but Davos is seen to have four and a half times as many Föhn days. This latter is a very notable preponderance, and one affecting not a little the pleasantness of a residence in an Alpine resort. To invalids each day of wind means a day indoors, for wind is far more trying than falling snow ; an invalid therefore would have, as regards wind, seven less days out of the 120 for outdoor exercise at Davos than at Arosa.

This is a result, I think, not to be unexpected from an investigation of the topography of the two places. Arosa clusters round two small and deeply set basins at the head of a curving and narrow valley, the lower end of which opens against high mountains. This is Arosa's only outlet, and faces north-east ; while throughout the rest of the circle it is completely shut in by the mountain masses of the Rothorn on the south, 9787 feet, with a splendid view from its summit ; the Weiss-horn, 8708 feet, on the north-west, and the Schiesshorn on the south-east. On the east is the Mayenfelder

Furka, a high and rugged pass of 8020 feet, running between the Furkahorn and the Amselfluh, but it is too high to be spoken of in any sense as an exposed point.

With Davos it is very different ; this lies right in the course of its valley, some third of the way down it, and only 240 feet below its summit ; while the lower or south-south-west end of the valley, instead of abutting against a lofty mountain mass, opens out into the broad vale between Lenz and Tiefenkasten.

This difference seems to me quite sufficient to account for the greater prevalence of the Föhn in Davos, and therefore enables me to believe that Herr Egger's observations on the prevalence of this wind are not only true for that particular winter, but are true generally.

**Mist.**—During the winter mist occurred only five or six times, and then lasted but a few hours.

**Hours of Possible Sunshine.**—In these shut-in valleys the length of possible sunshine is always short, though the amount of sunshine is great, for the sky is nearly always clear. This short duration makes every extra half-hour of extreme importance, and perhaps of even greater importance still is the time of day which receives the sunshine. Few invalids get out before nine o'clock, so that it matters comparatively little if the sun appear before that hour. With Alpine invalids the midday lunch is a substantial one o'clock meal. There is not much readiness, therefore, to go out for the afternoon walk before two ; hence the lengthening of the

afternoon sunshine by half an hour is of great moment. Thus it is not so much the earliness of the sunrise, but the lateness of its setting, which adds chiefly to the length of day enjoyed by the Alpine invalid.

At Arosa the hours are as follows :

	Sunrise.		Sunset.		Duration of Sunshine.
1885—Nov. 3rd	. 7.30	...	3.17	...	7.47 hours.
„ 14th	. 8.0	...	3.4	...	7.4 „
Dec. 2nd	. 8.21	...	2.40	...	6.19 „
„ 16th	. 8.46	...	2.19	...	5.33 „
1886—Jan. 4th	. 8.46	...	2.45	...	5.49 „
„ 15th	. 8.40	...	3.11	...	6.31 „
„ 30th	. 8.25	...	3.34	...	7.9 „
Feb. 14th	. 8.15	...	4.4	...	7.49 „
Mar. 2nd	. 7.59	...	4.21	...	8.22 „
„ 16th	. 7.52	...	4.52	...	9 „

St. Moritz :

Dec. 21st	. 10	...	3	...	5 „
Jan. 1st	. 10	...	3.5	...	5.5 „
Feb. 14th	. 7.45	...	3.50	...	8.5 „

Davos Platz :

Jan. 1st	. 10.3	...	3	...	4.57 „
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Davös Dorfli :

Jan. 1st	. 8.35	...	3.17	...	6.52 „
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Wiesen :

Jan. 1st	. 10.35	...	3.45	...	5.10 „
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Maloggia :

Jan. 1st	. 9.35	...	3.45	...	6.10 „
Feb. 14th	. 9.23	...	4.30	...	7.7 „

The hours of possible sunshine at the following places are—

	Arosa.	Davos Platz.	St. Moritz.	Wiesen.	Maloggia.
Nov. 1st	. 7.30	... 7.20	... —	... 7.20	... 7.10
„ 13th	. 7.2	... —	... 6	... —	... 6.45
Dec. 1st	. 6.20	... 5.15	... 5.15	... 5.15	... 6.30
„ 15th	. 5.35	... 5.5	... 5.5	... 5.6	... 6.15
Jan. 1st	. 5.52	... 5	... 5.5	... 5.10	... 6.6
„ 15th	. 6.31	... 5.30	... 5.20	... 5.50	... 6.20
Feb. 1st	. 7.10	... 6.20	... 7.5	... 5.15	... 6.50
„ 15th	. 7.50	... 7.45	... 8.5	... 7.40	... 7.8

From these tables it will be seen that, as regards the mere length of possible sunshine, Arosa is amongst the best, being equal with the Maloggia; but as regards the earliness of sunset it is worst, this occurring before three o'clock for six or seven weeks in the year. If English invalids wintered at Arosa, they would have to fall in with the different hours of sunshine and get up betimes.

Of the existing hotels in midwinter the Brunold enjoys most sunshine; somewhat less have the Rothorn, Waldhaus, and Seehof; whilst the Kurhaus has least: on the shortest days scarcely seeing the sun at all.

Arosa shares the following advantage along with the upper Engadine over Davos, Wiesen, Seewis, and other lower resorts—to wit, the snow melts later on in the year. This is due to its greater altitude, which is 800 feet higher than Davos Platz, 1400 above Wiesen, and just twice the elevation of Seewis. At the first appearance of snow-melting the resident at Arosa should flit to Wiesen or Seewis, where he would be sure to find the beauty of spring in its fulness, and so avoid most of the discomforts and problematical evils of this unpleasant period of the year.



If anything, more favoured in situation than Wiesen, though less as to accommodation and in less romantic scenery, lies Seewis, half an hour's drive from Pardisla, which is a village in the Prättigau four miles from Landquart. Either spot is about equally distant from Arosa. At present one would have to go by sleigh to Langwies, thence by diligence to Chur, and there change into the Davos diligence for Wiesen, or take the train to Pardisla if Seewis were selected. The distances are—Arosa to Langwies eight miles, Langwies to Chur thirteen and a half miles, Chur to Wiesen twenty-three miles and a half, Chur to Seewis from fourteen to sixteen miles and a half by rail.

In the matter of exercise Arosa as yet is not very suitable, the village paths are too steep and ill-kept for the gentle exertions of invalids; but potentially it is well off in this respect, for many beautiful walks with easy gradients could soon be made along the pine-clad slopes of the Tschuggen. A little wise and comparatively slight labour would enable it to outvie Davos in this respect, for there is an oppressive monotony about the latter which the charmingly varied outline of the hills around Arosa must ever prevent.

To reach Arosa the best way is by Basel and Zurich to Chur, thence by diligence to Langwies, and so by sleigh to Arosa. This sleigh journey could scarcely, I think, be anything but a rough one at the present, unless the long talked-of road be now made. But the way is short and beautiful, and rest would come at the end. The invalid should spend the night at Chur; or,

if unable to bear fatigue, at Langwies, where the Bär, though somewhat primitive, is roomy and clean, and possesses a landlady both capable and obliging.

If this road were made, the journey from England would be considerably easier than to either Davos or St. Moritz, consisting in a short twenty-mile diligence from Chur. Bäderer says in his 1887 Switzerland that it was to be made in 1887, but there was no sign of it in October of that year so far as I could see. Indeed, both at Langwies and Arosa I was told the inhabitants were if anything averse to it, for they made quite enough money in the summer season, and liked to have an idle winter in which to rest and spend their summer gains.

However, in 1887 Arosa was an impossible place for invalid wintering for any one unable to rough it to a considerable extent. The nearest medical aid, for instance, would have to be obtained from Davos, which is seven hours off along a toilsome footpath over the Strela Pass. But the possibilities of Arosa are great ; its climate is one of the best in Alpine Switzerland ; the great beauty of the spot is admitted by all, a beauty which one would get to love and feel fond of, not a beauty to oppress one ; the existing hotels are substantially built, and if under proper management would suffice quite well for the wants of a modest colony ; the late hours and evening entertainments of Davos would be avoided ; there would be no danger from overcrowding ; and as to a doctor, he would be sure to follow in the wake of patients.

With regard to drainage I am unable to speak, but I should imagine that each proprietor manages his own. This, if the colony grew to any size, would probably have to be altered, but there could be no difficulty in constructing a common drainage system ; and at present the individual arrangements are quite sufficient, for it must not be forgotten that Arosa has long been a favourite and much-frequented resort amongst the German Swiss. Indeed, Bädeler tells us to secure rooms in advance to avoid disappointment during the summer season, and that it is coming into favour as a winter resort. This last is exaggeration, for the Brunold alone keeps open after October, and five or six invalids is the largest number that has ever wintered there.\*

\* I have since learned from my friend Mr. Chavasse that a diligence now regularly runs the whole way from Chur to Arosa, and that the hotels remain open in winter. That there is, in fact, a regular winter season. Now that Davos has become a crowded health resort and St. Moritz tending to imitate its example Arosa is more than ever worthy of the consumptive's consideration (Dec. 1894).

## (C) THE CLIMATIC TREATMENT OF PULMONARY PHTHISIS

THE pulmonary phthisis concerning whose climatic treatment I am about to speak is caused by weakness, inherited or acquired, constitutional or local, which enables the bacillus of tubercle to multiply in the lungs of the individual so enfeebled.\* By an inherited weakness I do not mean you to understand that the bacillus itself is inherited—that pulmonary phthisis is congenital. Cases of extreme rarity show that this is possible, but they are so few that their consideration need not detain us here. Even the power of heredity in producing a special predisposition to phthisis has probably been greatly over-rated. Feeble parents will naturally produce feeble children, but it has not yet been satisfactorily shown that the children of tubercular parents must necessarily have a *tubercular* feebleness.†

\* Whether the bacillus can multiply in the lungs of perfectly healthy individuals is a moot question, not, so far as I know, yet answered. The weight of clinical evidence is against its doing so, at any rate, in temperate regions. The number of such instances must be very few, so that we need not hamper ourselves with this topic now.

† Nous n'avons recueilli aucun fait en faveur de l'hérédité de la phthisie. Louis, "Recherches de la Phthisie," p. 536.

Pulmonary consumption we may, then, consider to be a disease due chiefly to environment. This environment acts in two ways : it produces a physical weakness, and also a bacillus which can flourish in that weakness. Here, then, a great hope lights us : let us change the environment, and so do away both with its predisposition and the disease itself. In other words, climate is our all-powerful therapeutic weapon—the El Dorado of consumptives.

But when we scan the world's map to discover this ideal land, where is neither the bacillus nor even the shadow of tubercle, we grow sadder as we learn wisdom ; for wherever human beings are gathered together, there also is found this mighty enemy of their welfare. It is found amongst the snow and ice of Greenland as well as in tropical Brazil ; in the Alpine heights of Switzerland as well as on the hot sands of Egypt.

Yet, though we cannot send our patients to a climate where phthisis does not exist, still we can find one where it holds but a precarious tenure of the soil ; and to this task I will now address myself.

Statistics show that consumption varies directly with—

- (a) The density of the population.
- (b) The want of ventilation and drainage.
- (c) The confined nature of the occupation.
- (d) The want of open-air exercise.

Further, that with reference to the bacillus—

- (1) A warm climate is favourable to its development,  
as it has only as yet been cultivated outside



the body at temperatures varying between  $84^{\circ}$  and  $107^{\circ}$ .

(2) Moisture aids its multiplication.

(3) Stillness of the atmosphere assists it, for its growth occupies two or three weeks.

Lastly, as regards the individual affected, his chance of recovery depends chiefly upon—

(1) The functional activity of his lungs.

(2) His power of assimilating food.

(3) The energy of his vital processes generally.

(4) Residence in a temperature not widely differing from that of his birth.

Hence, our ideal climate must be one—

(a) With a very sparse population.

(b) Where the life may be largely an outdoor one.

(c) Where the houses have large and well-ventilated rooms.

(d) Where open-air employment may be obtained should employment be necessary.

(e) Where the inducements for physical exercise are great.

(f) Dry, sunny, and stimulating, especially stimulating to the organs of respiration and assimilation.

(g) Where there is free general movement of the air.

(h) Where good food can be easily obtained as well as the ordinary comforts of invalid life.

(i) Where the drainage is good.

(k) In temperature not greatly differing from that of England.

A mountain climate of from 5000 to 7000 feet approaches this most nearly.

(1) *Its Population is Sparse.*—Moreover, even for its sparseness, it has a low death-rate from consumption, which we may conclude denotes that the number of living consumptives is small. As there is some evidence to show that bacilli outside the body can increase in virulence, and light up fresh mischief in a phthisical convalescent who had become proof against the attacks of his own bacilli—further, as Bollinger has calculated that one phthisic can eject twenty million bacilli in twenty-four hours—the importance of having as few of these in the district as possible is obvious. The phthisical health-resort of Davos in Switzerland is a great sinner in this respect. Its winter population cannot be called sparse; and, as regards the condition of the hotels, the epithet crowded, in a phthisical sense, is a by no means unfair one. We must remember that this indoor condition is the most important one, as few phthisics spend less than two-thirds of their time within the house.

Denver in Colorado, though a populous city of 35,000 at the census of 1880, is in an open situation; its streets are wide, and its houses not closely packed. There is no great massing of phthisical subjects in hotels, as these are not merely for invalids. It is easy here, too, to live in separate houses or apartments.

Colorado Springs, seventy-five miles south of Denver, is still better off. Its population of 6000 is scattered

over four square miles, and the houses have good gardens around them.

In the Alps, Arosa and St. Moritz are much less crowded than Davos.

Of the Andean resorts I cannot learn enough to speak definitely on this point.

(2) *It is dry and sunny.*—During the winter, mountain health-resorts have very little snow and cloud. The atmosphere is one of perennial sunshine ; but the topography of the surrounding mountains greatly limits the duration of this, the average length of the day in the Swiss resorts being only six hours. The eastern slopes of the Rockies and the elevated plateaus of South America have a great advantage in this respect, their surrounding mountains being so far distant. But even with the limited day of the Swiss Alps these resorts have an immense superiority over our own in the matter of sunshine. What would we give to have six hours of cloudless sunshine in each of twenty-three days in January, as they had at Davos in 1887 ; or to possess the even more astonishing record of Denver, where, during five years and three quarters, there were only seventeen days when the sun was invisible throughout the whole day ! Compare Greenwich, where for fourteen consecutive days in November 1883, no sunshine was recorded.

Sunshine is of especial virtue in high altitudes ; for, owing to the greater diathermancy of the air, the difference of temperature between sunshine and shade is very great. Denison estimates that this difference

increases  $1^{\circ}$  for every 235 feet of altitude, so that at Denver (*e.g.*) it would be  $22^{\circ}$  greater than at the sea level. As the invalid is practically only out during sunshine, from a therapeutic point of view it would be fairer to judge of a high altitude climate by its sunshine temperature than by the usual method of shade temperature. So judged, it would come out as a warm climate, as mid-winter picnics testify ; and hence is explained its pleasantness, and the ease with which its apparent rigours are borne by the delicate ; for, within doors the patient has recourse to artificial heat. This penetrating power of the sun has another good effect. The sun rises, and it is day ; he sets, and night comes. There are no cold morning mists to be dispersed ; his warmth is upon you at once. Nor is there the grey of the twilight ; for he sets round-orbed and clearly, leaving only the darkness of night behind him. This makes our patient ready to get up betimes, and does not tempt him to linger out in the cooling moisture of the evening. I shall not dilate on the value of sunshine ; there can be no need to do so to any dweller among the dun cold mists of our cloud-ridden winters. One thing I will mention : there is nothing more inimical to the development of bacteria than bright sunshine acting on freely moving dry air containing plenty of oxygen.

This diathermancy of the atmosphere of high climates has one drawback : if it lets the heat through it easily in one direction, it must needs do so in the other. Hence, whatever has been stored up in the earth during the day is quickly parted with at night, making the

difference of temperature of mid-day and midnight very great. Thus, whilst the mean daily range of shade temperatures for the year 1874-75 at San Francisco was  $12^{\circ}$ , at Denver and Colorado Springs it was  $30^{\circ}$ —that is,  $18^{\circ}$  more. If we add to this the increased difference between sun and shade temperatures due to the height of Denver—viz.,  $22^{\circ}$ —we find that the difference between the temperature at mid-day in the sunshine and at midnight at Denver is  $40^{\circ}$  greater than the same difference would be at San Francisco.

But I fail to see that this is a great objection. The cold is a dry cold, and therefore felt with a comparative slightness. Moreover, as the patient is indoors, it can easily be counteracted. A greater danger is that of passing in and out of shade and sunshine during walks; but as this always exists, one soon gets into the habit of carrying a light woollen wrap and putting it off and on, much as we use an umbrella in England.

3. *It stimulates Respiration.*—The effect of this dry and rarefied air on respiration and on the lungs is matter of some dispute, and has been much investigated. It has been said that the air being rarefied, the lungs must needs take up a smaller amount of oxygen with each inhalation. But this seems very doubtful. Dr. Parkes states that in experiments on animals the amount of oxygen absorbed did not diminish so long as the percentage of oxygen in the air did not fall below fourteen. Now at 6000 feet the percentage of oxygen is sixteen, supposing that at sea-level to be twenty. At this elevation, then, the



rarefaction of the air would have no effect in lessening the absorption of oxygen. This tallies with our experience in climbing to those heights, for mountain sickness, the soroche of Peru, is not noticed till ten or more thousand feet have been reached.

Further, evidence is in favour of the statement that respiration, which is at first quickened, becomes deeper from a residence in high altitudes. In proof of this, we have the remarkably large chests so characteristic of dwellers in these regions.

Lastly, we have Frankland and Tyndall's experiments showing that candles burn quite as quickly on the summit of Mont Blanc as in Chamounix, and, moreover, that on the summit the combustion is more perfect, owing, as they think, to the greater mobility of the molecules of oxygen.

However these things may be, one clinical fact cannot be gainsaid—viz., that residence in the mountains increases the girth of the chest. This is perhaps the most satisfactory proof that the mechanism of respiration is stimulated. The dryness of the air enables it to absorb far more moisture from the lungs, and must tend to dry these when moist with morbid secretion. We find, accordingly, that râles and crepitations usually much diminish during a mountain residence.

4. *It improves Digestion.*—It is matter of common knowledge that elevation increases the appetite and both the powers of digestion and assimilation; that it enables one to undergo great physical exertion with little or none of the weariness which would accompany half the labour if performed at sea-level.

5. *Its Air moves freely.*—At these heights the movement of the air is largely untrammelled by the irregularities of the earth's surface ; hence, this movement is far steadier and continuous. In this relation mountains hold to lowlands the position that mid-ocean does to the coast-line. Though there may be as much movement, there is far less wind in the troublesome gusty sense we usually understand it. For this lack of wind Davos and Arosa stand out pre-eminent. Then come St. Moritz and Colorado Springs, while Denver seems to suffer considerably. We must remember that a little wind in a mountain health resort is far more trying than a similar one at a low level, owing to the great coldness of the air. This does not apply to the Andean health resorts, where the climate is much warmer, and their nearness to the Equator makes it much the same all the year round. For instance, Quito, at 9500 feet, is said to have an eternal spring with a never-ceasing vegetation under an ever-blue sky; and that central plain of Peru called the Montana, at 12,000 feet, which contains many cities and villages, has a similar climate. Perhaps Shakespeare had heard vague rumours of these when he wrote the Orpheus song in *Henry VIII.*, for in it are these lines :

“ To his music, plants and flowers  
Ever sprung : as sun and showers  
There had made a lasting spring.”

6. *The Comforts of Life.*—These are easily obtained both on the slopes of the Rockies and at the Alpine health resorts ; but on the Andes we are in Spanish South

America, and food and comfort are said to be far from an Englishman's ideal. Ventilation and drainage are well looked after both in Switzerland and Colorado ; but here, again, the Andean resorts are deficient. Suitable hotels and houses exist in all three.

7. *Employment*.—If we have to consider the question of a livelihood, so far as I know, the Rockies are our only satisfactory resorts. In Denver, a rising city and great railroad centre, with various manufactures ; much wholesale, retail, and banking business ; and unusually good educational opportunities, there is every chance of settling down. Wet Mountain Valley, 7500 feet, near Canon City, is known as the Briton's Paradise from the number of Englishmen who have settled in farms there. Here, too, is one of the richest mining countries of the world.

We have thus seen how nearly the mountain climate approaches our ideal one. It approaches it in another sense, in an almost, and some say an entire freedom from consumption in the native inhabitants. Thus, Dr. Archibald Smith says it only exists "as an exotic" in the Andes above 8000 feet, and yet large towns—*i.e.*, dense populations—exist at 9500 (Quito) and 13,500 (La Paz). It has also this great advantage : it can be continuously lived in, not merely during the winter season. Its chief drawback is its inaccessibility and distance from centres of human thought and action. Colorado is best off in these respects. Denver has the stir of a business centre, while the small town of Colorado Springs mostly consists of cultured

people who have settled there for the sake of their health.

I will finish my description of mountain climates by reading to you the astonishing history of Dr. Guilbert's Swiss clockmaker, as related by Dr. Theodore Williams.

A Swiss clockmaker, aged 27, who had lost both parents from consumption, was attacked with cough and large hæmoptysis, followed by loss of flesh and night-sweats. He was recommended a change of climate, and accordingly repaired to America and set up a watchmaker's shop at Panama, a low-lying town situated on the isthmus of that name. Here the disease progressed ; and, following advice, he travelled to Quito, in the Andes, 9500 feet above the sea-level, where he remained six months, at the end of which period he had regained his original weight, lost the fever and night-sweats, and had an excellent appetite and scarcely any cough. He returned to Panama, and led a temperate and moderate life ; but after some months the cough, night-sweats, and fever all returned, accompanied by diarrhoea and wasting. A second visit to Quito was recommended, and carried out with the same favourable results, and a return to Panama with the same unfavourable ones. A third journey was then made to Quito, where he remained nine months, and was strong enough to visit some of the higher mountains of the range, though it is not recorded that he performed any mountaineering feats. He returned to Panama so well that he had a firm conviction that he was cured ; but he never lost his cough, and a recurrence of his symptoms soon drove him again to the heights. This time he chose Arequipa, a town lying between 8000 and 9000 feet above the level of the sea, and here for the fifth time he regained his health ; but, with a strange fatality, he quitted the mountains and retired to Tacna, a place at a considerably lower level. Some time afterwards Dr. Guilbert examined



him, and described him as in a state of terrible emaciation, looking more like a corpse than a living being, suffering from fever and night-sweats, with entire loss of appetite, diarrhœa, and a troublesome fistula, and having severe cough and large expectoration. He also detected the existence of cavities in both lungs, and scattered crepitation at the bases; and having prescribed palliatives, he quitted the patient under the impression that he should see his face no more. But he was mistaken. Dr. Guilbert ascended to La Paz, the capital of Bolivia, at an altitude of 13,500 feet, and here shortly afterwards the patient turned up, having followed his doctor more slowly, and now astonishing him by his rapid improvement. The fever, sweats, diarrhœa, had all disappeared, the expectoration had diminished, the appetite returned; and during some excursions they made together, the patient is reported to have walked as well as the doctor, and to have been free from the soroche or mountain sickness which is troublesome to new comers in these parts. The cavities remained unchanged. Thus, for the seventh time, the Andes rescued him from the jaws of death; and it had been well if he had clung to them. Unfortunately, he returned to Europe and Neuchâtel on private business; the disease made fresh progress. He was ordered to Eaux Bonnes, and died there a few days after his arrival.

Next to the climate of high altitudes comes that of **dry hot lowlands**, which also has many excellent points. Its inhabitants are sparse; its atmosphere is dry, clear, and sunny; there is less difference between day and night temperatures and between sunshine and shade than in high altitudes, though this difference is still considerable. It has, too, the great advantage of a longer day between sunrise and sunset, and hence more time can be spent out of doors. But its climate is by no means so bracing, there is not the same inducement



to physical exertion, nor is this so easily borne, and the chances of obtaining remunerative employment are small. Except at Cairo and its health resort Heluan, or during life on a Nile boat, the comforts of life and good food are hard to get, and at these living is very expensive and, moreover, can only be indulged in during the winter—five months of the year. In Cairo the drainage might be better, though the house accommodation is good. A great objection to Cairo is the strong temptation to indulge in the indiscretions of social gaiety which it affords.

The typical life in dry hot lowlands is tent life in the desert ; but to follow this we must take unto ourselves again the life of Esau, but with this great drawback, which Esau had not—to wit, we must forego the society of our fellows. This has been always too severe a trial for an invalid to permanently face, and sooner or later he has returned to civilisation.

Next best to this is perhaps the wild, lonely, pastoral life led on the great inland plain of Australia which goes by the name of the Riverina. It lies chiefly in New South Wales ; its southern boundary is the river Murray and Victoria ; and on the west is the great central Australian desert. It consists of rolling prairies, destitute mostly of tree and grass, but producing vast quantities of the salt bush which affords such excellent fodder for sheep. Life is lived in the saddle, for your nearest neighbour is twenty to thirty miles off. Hot winds and dust storms are frequent, but in the summer, days of still, cloudless sunshine are the rule. The

summer heat is great but dry, the daily range of temperature rather large, but sudden fluctuations of this are infrequent. In the winter there is a little morning frost. But for the occasional storms and the rather too great heat of summer, this seems to be an ideal climate for consumptives—one, too, where a man of fair physique could earn his livelihood. But its chief virtue—its distance from the busy haunts of men—is at the same time its chief drawback ; and, though I have often spoken highly of it, I have never yet succeeded in inducing a patient to travel thither.

The climate of dry hot lowlands is, then, almost as dry and has more sunshine than Alpine resorts ; it is as free from wind, and its air is as pure. But it lacks some of the stimulating effect on respiration and appetite, nor is it socially so pleasant.

Another form of health-giving climate is that enjoyed during an **ocean voyage**. I shall only discuss the Australian voyage, and this when made in a sailing vessel. Here the traveller has plenty of elbow-room below deck, and the food is good, and he is free from dust and noise. In the north-east trades he can remain on deck the whole day in brilliant sunshine, the moisture of the air soothes his cough, whilst the stimulating action of the salts it contains braces his appetite. But in the Calm Belts and on the Equator the excessive moisture and motionless air induce a sense of oppressive languor and, below deck, almost unbearable closeness. With the advent of the south-east trades we come into our delightful climate again ; and thence to Melbourne,

through the boisterous west-wind region, the weather is usually cool, bracing, and healthful ; though caution must be shown for fear of chill. Such a voyage suffers from insufficient ventilation and smallness of rooms ; also from want of inducement to take exercise. It is a tonic to the digestive but not to the respiratory system, and, as a rule, it soothes a worn-out nervous system.

Lastly, we have the climate of **warm, moderately dry sea-boards**. Of these more has been written than of any other kind of health resort. They are more familiar to us than any others, and I shall therefore not describe them in any detail. Their population cannot usually be termed sparse. They have a longer day than the mountain resorts, but they cannot be credited with so great a proportion of sunshine in their daylight, nor is their sunshine so penetrating ; neither is their air so dry nor so stimulating to the lungs. Their scenery is not so grand, but more loveable and charming ; and it is pleasanter to sit under orange trees than tramp over snow. Socially, their life is of the pleasantest and travel between them very easy ; therefore they are much sought after, and hence employment is apt to be far less than the demand for it. Their accommodation is of the best and very reasonable, but they are not fit to reside in during summer. These remarks refer more particularly to the French and Italian Rivas and the Canaries. In southern California you can stay all the year round and, as yet, it is possible to obtain a livelihood there. It, too, is least troubled with winds, and has the smallest daily and annual range of temperature.

In all these various sanatoria have all varieties of consumptives benefited, and in them all have they all similarly got worse. But evidence goes strongly to prove that inflammatory cases do best in the mountains ; that cases with little energy and resistance improve most in Egypt ; and that for those who have fallen a prey to the disease from overwork or continued anxiety, an ocean voyage is the best recuperative agent.

By inflammatory cases I mean all those resulting from unresolved pneumonias or pleurisies, or from irritative employments—dust phthises. That these should be benefited by that climate which especially stimulates the respiratory organs is only what we should expect. The increased lymph and blood circulation removes old inflammatory deposits, and the deeper respiratory movements stretch old adhesions. We must not forget that these are the cases which do best in any climate, as the onset of phthisis has accrued from a more or less accidental evil, and not from an inherited weak constitution.

Chronic tubercular phthisis also does best in mountain climates, no doubt owing to the great increase in all the functions which results from this residence, which enables the body to make a better fight against its, in this case, sluggish enemy.

In fine, those who have sufficient energy and vitality to take advantage of the virtues of high altitudes had better go there ; those who are feeble, fragile, and excitable, to Egypt. Ocean voyages are not often to be recommended to women ; and the Australian Riverina

is at present evidently adapted to very few ; though one cannot see why a colony of invalids should not be started at some spot in it, just as such a colony has been established at Davos.

I have left the seaboard of the Riviéras and southern California to the last because, as climates pure and simple, I think them least effective. Still, three of my patients who have gone to California have done excellently ; and we all have good things to say of the Riviéras. But this is because I think they are so easy of approach and life is so pleasant—two factors which in many cases more than counterbalance their less valuable climate.

Finally, we must never forget that no case of acute phthisis, nor any in the active third stage, should leave our shores. As patients learn to come to us earlier in their illness, and we correspondingly become abler in our diagnosis, so will the question of dealing with these advanced cases become more and more a thing of the past, and consequently treatment by climate, the results of which are so extremely satisfactory in first-stage phthisis, will become more and more popular.



## V. NON-TUBERCULAR PULMONARY CAVITIES

IN this paper I purpose speaking of the origin and treatment of cavities in the lung produced otherwise than by the bacillus of tubercle. My paper naturally divides into two parts—the former relating to ætiology and the latter to treatment.

First then, as to the causes : they may be grouped under these heads :—(1) Inflammations originating within the lungs ; (2) Inflammations originating outside the lungs ; (3) New growths ; (4) Parasites ; (5) Injuries ; (6) Emphysema.

(1) Of **Inflammations arising within the Lungs** perhaps *Pneumonia* should occupy the first place. Here the primary outburst may be of so acute a nature as to proceed straightway to purulent disintegration, or, and this more commonly, the greyly hepatised lung fails to resolve, and gradually transforms into pus. The former is the more hopeful variety, but Jurgensen states that the number of reliable observations are too small to form a positive opinion. Laennec, for instance, says that out of twenty cases he had only two deaths ; whereas Huss lost twelve out of his twenty cases.

The latter form is well exemplified in a private patient whom I had the opportunity of watching for nearly two years. One year before I saw him, when in a debilitated condition, he contracted an acute pneumonia which confined him to his room for two months. He continued an invalid for seven months longer, when one morning he suddenly expectorated a large quantity of extremely foetid pus. Five months later—that is, fourteen months from the onset of the pneumonia—I first saw him; he then was bringing up some ten ounces of thin sero-purulent offensive matter, three or four ounces at a time, with fits of most violent coughing. I found his right base to be deficient in resonance, movement but little lessened, breath sounds somewhat bronchial with large sticky crepitations. Here were no signs of lung cavity, but as Drs. Symes Thompson and Theodore Williams, who sent him to me, had both informed me of the existence of such, I concluded that they were at present filled with secretion, as indeed afterwards proved to be the case. At the extreme left base crepitations were also heard, and this, in my opinion, negatived operative procedure concerning which he had consulted me. His condition varied considerably, sometimes improving for months together, and it was only during the last week of his life that he was entirely confined to his room. He died with subacute septic symptoms.

But cavities may result from *Broncho-pneumonia* as well as from the acute fibrinous form. Such is the case recorded by Douglas Powell and R. W. Lyall in *The Lancet*, vol. ii. No. 1, 1880.

A man, 49, had bronchitis in December 1878, and in February 1879 pleuro-pneumonia with foetid expectoration. He improved and then relapsed, so that in July 1879 he was admitted to Middlesex Hospital. The lower lobe of the right lung was consolidated with a central excavation at the level of the angle of the scapula. There was hectic and diarrhoea, with about one pint of expectoration in the twenty-four hours. The lung was incised and drained with almost complete cessation of cough and expectoration as an immediate result. But in about three weeks some return of foetor in the breath and discharge occurred, and he died fifty days after the operation from pleuro-pneumonia of the opposite lung. At the post mortem several intercommunicating cavities were found and the lobe was firmly adherent throughout. The drainage tube passed through the eighth space into a contracted cavity which opened into the main cavity by a short dilated bronchus. The tube-containing cavity in contracting had partially obstructed the end of the tube, thus causing insufficient drainage, with death probably from septicæmia.

The various varieties of non-tubercular *Fibroid Phthises*—such as miners' lung, grinders' rot, syphilis—generally produce, sooner or later, bronchiectatic cavities; these usually, but not always, occur at the base; they are very slow in progress and have but small tendency to enlarge by excavating the lung parenchyma; their walls are smooth and, to some extent at any rate, lined by bronchial mucous membrane. Perhaps the most painful symptom connected with these cases is the extreme foetidity of the sputum.

A case in point is that of a man admitted into Brompton Hospital under Dr. Williams with his right lung riddled with

bronchiectatic cavities, and quantities of intensely foetid expectoration which came up with suffocating paroxysms of coughing three or four times a day, at which times the atmosphere of the passage outside the door of his ward was almost unbearable, notwithstanding every effort at disinfection. The man's condition was so disgusting that surgical interference was at once sought, and Professor Marshall incised in the fourth right space just without the nipple, over what appeared to be the largest cavity, and inserted a drainage tube into a cavity the size of a duck's egg. The result was astonishing; the sputum diminished one-half and the foetidity almost entirely vanished, while the discharge from the cavity was but slight. But the man died three weeks later with septicæmic symptoms.

In the absence of the pathologist the post-mortem examination was made by myself. I found the right pleura almost entirely adherent, especially in the latero-anterior region where the bronchiectases chiefly lay. The drainage tube passed straight into the outer and lower portion of the largest cavity, which was at least double the size of any other. This it completely drained. There was very little retained pus in any cavity, and what there was had no special foetor. The lung was greatly fibrosed throughout.

In the cerebellum, centrally situated with its long axis transversely horizontal, was a cavity the size and shape of a duck's egg containing odourless pus and lined by a wall at least one-sixteenth of an inch in thickness. This was evidently of long standing and probably quite unconnected with the lung mischief. The cerebellar tissue around it and the brain substance generally, showed no signs of inflammation or disorganisation to a naked-eye examination.

This case is interesting as an instance of the advantage of chronicity in rendering the operation of pneumonectomy a comparatively trivial procedure owing to the

firm adhesion of lung and chest wall. It is also noteworthy that the drainage of one cavity—and that one by no means the lowest in situation, neither when the patient was supine nor erect—practically did away with the fœtor. To those of us who have witnessed the infinite distress caused to both friends and patient by this symptom, this fact must call for serious consideration and make us debate whether external drainage is not justifiable for the relief of the putridity alone.

*Foreign Bodies* inhaled through the larynx are not an infrequent cause of pulmonary cavity; these naturally occur nearly always in one or other lower lobe.

Dr. Strange, of Worcester, recorded an interesting case in the *B. M. J.*, vol. ii. 1887, p. 1145. The patient was a woman, 23, of fine physique, but with a history of scrofulous abscesses of hands and feet in infancy. On February 17, 1887, several teeth were extracted under chloroform and the account of the operation given Dr. Strange by a friend who went with her to the dentist left no doubt in his mind that a stump of a tooth had gone into the trachea. A few days afterwards a troublesome cough arose with muco-purulent expectoration; these symptoms continued till June 15th, when she was admitted to Worcester Infirmary. She had never been completely laid up, nor had she lost much flesh, but her strength was failing and her cough and expectoration increasing. Some rings of cartilage, horribly offensive, had been found in her sputum. Her cough was now almost incessant, and she spat up two or three pints of stinking expectoration, containing broken-down lung-tissue, daily. She could not lie down, and had bed-sores. There was a hectic varying from 101·4 to normal. On the right side there were coarse râles from the upper third of the scapula to the ninth rib posteriorly, with



dulness, as well as tenderness over the third, fourth, and fifth ribs anteriorly, but with no indication of pointing or bulging. The condition of the rest of the right lung and the whole of the left was fair. On July 18th Dr. Strange inserted an aspirator needle in the ninth space one inch posteriorly to the axillary line; a drop of pus appeared. Thereupon a large curved trocar was pushed in eight inches towards the fifth interspace in front. Most offensive pus now came in large quantities; a drainage tube was inserted through the cannula of the trocar and the cannula then withdrawn. The dressing was carbolic tow: there was no escape into the pleura. In a few days the gangrenous odour was quite gone and the expectoration became very slight. September 12th she was discharged cured. On October 29th she had gained two stones. Her right lung appeared to be normal except for some dulness at the base. There was some dyspnœa and irritative cough on exertion.

This almost complete recovery from such a terrible condition illustrates forcibly the immense defensive power which healthy lung-tissue has, and enables us to understand why it is that the bacillus of tubercle is apparently so selective in its choice of victims.

(2) **Next, concerning Cavities due to Inflammations arising outside the Lungs.**

*Empyema* is by far the commonest cause of these.

In this connection the case of a gentleman I saw in 1886 with Dr. Bernays, of Solihull, is of interest. On May 3rd he took cold at a wedding; on the 6th Dr. Bernays saw him and found crepitations at both bases, but these were more marked on the left side, where there was also dulness and tubular breath sounds. T. 103°, P. 120, R. 30 (8 P.M.) On the 7th

one-third of the left lung appeared to be solid. On the 9th the breath sounds were fainter and only slightly tubular. On the 16th the left chest was dull as high as the scapular spine, and over its lower half slight œgophony was perceptible. The temperature, which on the 9th had fallen to  $99^{\circ}$ —marking, apparently, the crisis of the *pneumonia* portion of the illness—slowly rose thereafter, with evening exacerbations of  $2^{\circ}$ , till it reached  $103^{\circ}$  again on the 24th, on which day a profuse sweat occurred. The sweating continued, and on the 26th the sputum became purulent, with a curiously offensive odour. This condition persisted till the evening of the 30th, when three or four ounces of very fœtid pus was expectorated. I saw him with Dr. Bernays on the 31st, and found moist friction sounds with feeble sub-bronchial breath sounds throughout the lower half of the left chest. Although no pus could be withdrawn by an exploring syringe inserted in three situations, yet the diagnosis of “empyema following pleuro-pneumonia and rupturing through lung” was quite clear. As fœtid pus in small quantities was still being coughed up and the patient’s general condition a very serious one, immediate external drainage was advised. This was refused, and a week’s grace requested, at the end of which time he was better in every way—temperature lower, expectoration less, sweating almost gone, dulness lessened—though sticky crepitations, indicative of the gliding of two lymph-coated pleuræ against one another, were still heard at the base of the lung. I again advised external drainage, but now far less urgently. The outlook either way seemed long and troublesome. He was 54, a chronic bronchitic, with considerable emphysema and granular kidneys. If he were drained the sinus would be probably months in closing and there was a chance that he might continue to go on improving as at present; so I did not press my advice. Two weeks later I saw him again and never was I more thankful that my advice had not been taken. Though still feeble and occasionally

expectorating somewhat offensive material he was wonderfully improved, was in fact convalescent, and a month later Dr. Bernays could find no sign of evil import except that of slight deficiency in the percussion note. I saw him in 1889 returning from business as brisk as a bee and looking the picture of health. This was evidently an instance where Nature did better alone than she would have done with surgical aid. It is an instance, too, of a progress which one not so rarely watches in children, but in an adult, unfortunately, most seldom, and, in a man such as our patient was, one which should barely be admitted as possible when discussing treatment.

Another cause is a *Chronic Pleurisy* which may slowly invade the lung, producing fibrosis of this with consequent bronchiectasis, or else fibroid material which is very unstable and apt to break down. This is not an uncommon condition ; to a slight extent it obtains after most pleurisies of long duration, though far less often observed now than in former days when patients were allowed to go about with fluid effusions, for whose removal no attempt had been made. I remember one well-marked case under the care of Dr. Rickards, where the pleura was a quarter to half an inch in thickness and the neighbouring lung quite carnified and riddled with dilated bronchi. In such cases the cavernous condition seldom calls for treatment as the lung has previously become practically useless, and the expectoration is scanty or absent.

*Subphrenic Abscess* is a not uncommon cause of lung cavity from rupture of its contents through the diaphragm and the adherent lung into a bronchus. These are perhaps oftenest due to abscess of the liver,

but they arise also in the course of gastric and duodenal ulcer, perityphlitis, abscess of the spleen, and purulent peritonitis occurring from other causes. A well-marked instance of causal *perityphlitis* is a case I had charge of when acting as house-physician at St. Thomas's. A man was admitted expectorating pus and died soon after. The whole of his right lumbar region was one large sinus of pus bounded internally by the colon. This sinus led to a large subphrenic abscess, and thence into the lung, a large portion of the lowest lobe of which was converted into a ragged abscess cavity.

*Abscesses of the Spleen* are more apt to occur in malarial countries than in England; yet, even in Bengal, Dr. Bosanno Chowdhoo, who relates the following case, states that out of 30,000 cases of malaria which he had personally treated, he only saw three with splenic abscess.

One of these was a man of forty, who for some months had had a swelling in the splenic region with occasional pyrexia, and lately had spat up blood and pus. The spleen was found to be large; there were crepitations in the left lung, and the right lung was normal; the cough was only slight (and hence, says Dr. Chowdhoo dogmatically, it could be neither phthisis nor bronchitis). Abscess of the spleen was diagnosed, and gallic acid, gr. 5; ac. sulph. dil., ℥ 10; extr. ergot liq., ℥ 20, was given thrice daily. The expectoration gradually lessened, but after a while "pointing" was observed in the splenic region; the abscess was then opened through the abdominal wall, 10 ozs. of pus and some splenic debris came away, and fourteen days later the man was discharged cured.

*Pyæmia and the Specific Fevers* not seldom produce

lung cavities, but these are often only ante-mortem occurrences and multiple, so that little can be done for them.

Dr. Macgregor records a typical instance in the *British Medical Journal*, ii. 1887, p. 1219. A boy of 10 fell into a sewer; for ten days thereafter he had malaise, and for fourteen more typhoidal symptoms; then congestion of the lungs came on, with, four days later, pulmonary gangrene and left pneumothorax. Three days after this he died. The temperature had varied from  $96^{\circ}$  to  $106^{\circ}\cdot8$ . The right lung was the site of many circumscribed abscesses, from the size of a pea to that of a walnut. The left lung was collapsed, and towards the axillary base a small punched-out perforation from the giving way of a necrotic abscess was present.

But that we should not always regard such pyæmic abscesses as hopeless I think the following case shows.

Some time ago I saw in consultation with Dr. Wade, Mr. Taylor, and Dr. Ward, a lady who six weeks previously when at the theatre was seized with alarming uterine hæmorrhage, for which a sponge tent was temporarily inserted. The next day this was removed, and the uterus swabbed out with perchloride of iron. A few days later one or two severe rigors occurred, and a tumour was felt by vaginal examination on the left side of the uterus. Mr. Taylor therefore explored, and found a collection of reddish serum on the left of the pelvis, which was held in this situation by adherent omentum. He liberated this, cleaned the pelvis, and removed the appendages. There was considerable improvement for about ten days, when the rigors returned, and the temperature assumed a decidedly septicæmic character and signs of lung involvement arose. When I saw her later on there was considerable dulness—but by no means absolute—up to the level of the eighth rib in the mid-axillary line on the



left side, this dulness joining the heart dulness anteriorly and reaching round, at a somewhat lower level, over the posterior, surface of the left chest. Friction and crepitation sounds were heard over the whole of this dull area, the crepitations also extending into the axilla and above the nipple. There was a very troublesome dry cough, and the breath had constantly the fœtor of retained pus to a slight degree. The breath sounds were feeble over the whole lower lobe, but nowhere absent nor bronchial. The right lung appeared healthy and there was no indication of mischief elsewhere. The diagnosis at best could but be a doubtful one. That pleuro-pneumonia of a low type, with septicæmic symptoms, existed was evident. The friction sounds heard over the dull area negated an accumulation of fluid, yet pleuræ coated with a low form of curdy lymph might perhaps have accounted for the whole condition. But the peculiar fœtor of the breath and its accompanying dry cough pointed somewhat strongly to pulmonary abscess, and the balance of opinion lay in that direction. The localisation of the position of the abscess to any particular part of the lower lobe was an impossibility, though for my own part I certainly expected it to be in the dull area, and indeed hoped to find it by a puncture through the eighth space in the mid-axillary line; but at the post mortem I found a cavity the size of a large walnut occupying the extreme apex of the lower lobe and filled with grumous material; this corresponded to the *posterior* axillary line and the eighth space—that is, well above the line of dulness. Below this, the lower lobe was in a condition corresponding to the prune-juice stage of pneumonia. There was some lymph on the pleura, but otherwise the chest organs were fairly healthy and the only other evidence of pus was a few drops on one of the ovarian stumps in the pelvis.

This abscess was decidedly circumscribed, had indeed a definite wall to be incised, and I cannot help believing that if it had been opened during life there would have been a fair chance of saving the patient's life.

(3) **New Growths.**—I need say little concerning carcinoma and sarcoma; whether they break down and form cavities or not makes but little difference to the treatment.

(4) But it is very different with regard to **Parasites.** One would think an *hydatid cyst* if recognised before its rupture and evacuated should give as good result in the lung as in the liver. But the reported cases are as yet very far from attaining this favourable position. And if treatment has to come *after* rupture, affairs are much more gloomy.

A man, between 30 and 40, whose brother died in the General Hospital of suppurating hepatic hydatid, was an out-patient of mine for over three years, and was also under my care inside the hospital and at the Jaffray Hospital. When he first came he had already had lung symptoms for four years, evidently referable to hydatid disease. Both his lower lobes were full of crepitations, there was considerable dulness and the breath sounds were feeble. His expectoration, apart from the cysts, several of which he brought up on the average once a month, was purely bronchitic and due to the irritation set up by these; but latterly it has become muco-purulent and at times there are signs of small cavities in both lungs, the upper lobes being now also involved. His two great troubles are an exasperating cough and constantly increasing dyspnœa, the mal-oxidation resulting from which is slowly enfeebling him and degenerating his tissues generally.

Though his sputum was several times examined for bacilli, and though he was lying alongside phthisical patients for months together at the Jaffray Hospital, yet these were never found. Such cases give one cause for reflection. Lung-tissue more hopelessly *hors de combat* as to resisting an invader can scarcely exist. Yet in the long course

of seven or eight years the invasion never occurred. This surely is a testimony to the pre-eminent importance of the factor, heredity.

(5) **External Injuries** involving the lung may produce cavities. Schneider of Königsberg has successfully resected a rib and portion of lung for gangrene of the latter produced by a gunshot wound. But the conduct of these cases is almost entirely surgical and I will do no more than mention them.

Lastly, the bullæ of **Emphysema** occasionally arrive at a size which not improperly enables them to be designated as cavities. They are not without importance, for the rupture of a bleb may lead to sudden pneumothorax in an otherwise perfectly healthy individual. It is by no means necessary for this rupture to occur only at times of exertion. There was a patient at the General Hospital six years ago, under the care of Dr. Wade, who met with this accident whilst quietly pursuing his avocation in a sitting posture ; this perhaps accounts for the comparative mildness of the onset, as the rupture was not enlarged by forcible respiration. His family and his own personal history were excellent. He had done a good deal of professional running, and this may have been the cause of the emphysema. The prognosis is generally good in these cases.\*

\* I have now (December 1894) for the last six months been seeing with Mr. Mann a gentleman of 58 who woke up one morning at 4 A.M., turned sharply over in bed from right to left, and in doing so felt a sharp pain beneath his manubrium, shooting towards the left, severe enough to keep him awake some time. He got up as usual

In considering the *treatment* of these cavities I will first point out the results of Nature's unaided efforts at cure, and the dangers attending these.

Jurgensen, speaking in general terms, states that recovery is not so very uncommon—this he says in 1876, before their surgical treatment had entered the range of practical medicine, for the first recorded case of this is, I believe, that of Radek in 1878 (*Centralblatt für Chirurgie*, No. 44, 1878, p. 750). He thinks the prognosis, however, decidedly unfavourable, if the cause be due to infective emboli, putrid bronchitis, bronchiectasis, and sometimes also after acute pneumonia. It is, too, much more unfavourable when the upper lobe is attacked : this is evident for two reasons—1st, the lower lobes are very likely to become diseased from overflow and gravitation of discharge into them ; and 2nd, the upper chest lends itself much less readily to the contraction of lung necessary for the healing process to take place. In the 7 cases reported by Leyden and Jaffé, 3 were in the upper and 4 in the lower lobe : the 3 upper all proved fatal. Of the 4 lower, 2 were completely cured, 1 incompletely, and

at 8 A.M., but perceived his breathing to be not so good, especially when going upstairs. When I saw him the following day there was evidence of pneumothorax over most of the left chest, though the right lung and heart did not appear displaced. Slight double dry pleurisy supervened, but the cavity has steadily contracted and its signs have now completely vanished. With a perfectly good previous history, an unusually young and healthy-looking man, no lung signs, and good recovery, emphysema appears to me to be the only possible cause.



the 4th was progressing favourably at the date of the report.

But imperfect recovery is, after death, the commonest result if Nature be left to herself : a pyogenic cavity or cavernous system forms, by which large quantities of pus are daily secreted. The system may withstand this for years—two to ten or fifteen—but sooner or later it breaks down under the exhausting drain, and death results much as it does in cases of unhealed psoas abscess—with lardaceous and fatty degeneration. But other dangers also exist : the anatomical structure of the lungs and their functional activity lend themselves greatly to auto-infection, and also to infection from without. Finally, their disease lessens the oxidation of the blood, and hence of the tissues generally, thus diminishing the vitality of the whole organism and laying it especially open to pathological degeneration and to the inroad of germs.

The question of *tubercle* thus arising in previously thoroughly sound organisms is one of great interest and must be my apology for quoting the two following cases :—

The first occurred in the practice of M. Varnier, and is reported in the *B. M. J.* for 1887, vol. i. p. 351. A male, previous and family histories excellent. Three days prior to admission he complained of stitch in his left side. There was some dulness at the posterior base, but no fever. Eighteen days later this dull area was punctured and hooklets withdrawn. The next morning he expectorated hydatids with clear liquid and blood, whilst hydropneumothorax developed. The chest was then opened, but nothing escaped, though the next morning



hydatid débris was seen on the dressing. One month later he died.

The post mortem showed a hydatid sac at the base of the left lung, which was separated from the chest wall by only a thin portion of lung. In both lungs there was recent miliary tuberculosis, which M. Varnier suggests was due to infection through the wound.

On the other hand, Professor Gairdner showed at a Glasgow Society in January 1886 (*B. M. J.*, i. 1886, p. 21) the lung of a man, aged sixty-six, who had frequently been under treatment at the Glasgow Infirmary with symptoms of bronchitis and emphysema accompanied by considerable, probably bronchiectatic, excavation at the left base, which, however, added but little to the gravity of his symptoms. The patient stated that he had been very liable to bronchitis since an attack of "inflammation of the lungs thirty years ago." He had had three wives, all of whom had died of phthisis or scrofulous disease. By these, sixteen children had been born to him, of whom but one, the first child of the first wife was alive in 1883. He had therefore been excessively exposed to tubercular virus.

At the post mortem there was one largely dilated bronchus and many smaller ones, but *nowhere was there any deposit of tubercle.*

That the bacilli of tubercle *do* exist in these cavities there can be no doubt, for they have been found in the expectoration as well as in the cavities themselves. In the sputum of a gentleman suffering from double bronchiectasis (*vide* p. 253) I found a few imperfect bacilli as well as some spores. Indeed, as Dr. Filleau, in an able monograph on the treatment of phthisis, remarks: "Bacilli may be found even in strong and healthy individuals—that is to say, in those who exhibit

no tubercular taint. They are constantly found in those who live long in close proximity with tubercular subjects. To cite only one example, we have repeatedly examined our own sputa, and have regularly observed in them several bacilli and a few spores. We even preserve a very remarkable slide on which is seen a large epithelial cell closely resembling a giant cell, inside which are five undoubted bacilli. The preparation was made by Ehrlich's method, and is of so typical a nature that one could swear it came from a tubercular patient. However, it was furnished by one of us, of whose health," he naïvely goes on to say, "there can be no shadow of a doubt." In fact, there can be no doubt that bacilli glide in through our vocal cords along with many other impurities, but luckily, as a rule, only to find themselves on a barren and sterile soil, in which they live with much the same kind of vitality that frogs exhibit when they dream away our long inhospitable winter—a winter to them barren and unfertile, in which they neither marry nor are given in marriage. But—and this is the point to remember—the bacilli are there, ready to revivify themselves should the soil at any time change to a more kindly nature. Further, the tubercle bacillus has a great persistency of life; for MM. Chantemesse and Widal (*Archives de Médecine Expérimentale*, i. 30, 1889) have found it capable of reproduction after fifty and seventy days' existence in sterilised Seine water. Tubercle cannot arise without bacilli, and yet these are harmless in an unfertile soil. Bacilli we have always with us, hence the

grave importance of denying them any fertility of soil.

*Bis dat qui cito dat* must then be the motto of all our efforts. We all know how almost interminable is the course of a one-sided fibroid phthisis. And how is the end at last reached? "In most cases," says Douglas Powell, "sooner or later the opposite lung is affected and *generally with grey tubercle*, unless an intercurrent disease cut off the patient." As Hilton Fagge has well said, "Tubercle is the product of a modified inflammation." It is our duty then to do what we can to prevent this modification taking place.

But though I have spoken somewhat despondingly of its power if left alone, yet we must not forget the virtue of the *vis naturæ* residing in each one of us. After all, even in the matter of tubercle, it is but a question of degree : the physiological fades imperceptibly into the pathological. With some of us the bacillus would never have the ghost of a chance ; with others, the first break down of lung-tissue would enable it to win an easy victory ; while the bodies of most of us lie between these two.

The means at our disposal for the treatment of lung cavities are the following :

- (1) Calisthenics ;
- (2) Exhibition of drugs by the mouth ;
- (3) Exhibition of drugs by the trachea ;
- (4) Exhibition of drugs by subcutaneous injection ;
- (5) Exhibition of drugs by their injection into the cavities themselves ;

(6) Incising the cavity and draining it—1st by an ordinary tube, 2nd by perflation ;

(7) Resection of the diseased lung.

(1) **Calisthenics** (“ exercises for health, strength, or elegance ”) are naturally of service only when an outlet for the discharge of the contents of a cavity has already been formed. But as it is extremely rare for even a single cavity to caseate and dry up without rupture into a bronchus, this limitation is not of much importance. They are of use in these ways :—(1) To increase the movement of the lungs, thus increasing the oxidation of the blood, and so tending to obviate the fatty and lardaceous degeneration of the body generally, and to improve the circulation and vitality of the lungs themselves ; in fact, to give them the advantages of *passive* exercise. (2) By placing the body in a suitable position they enable the cavities to drain better. Thus, Dr. Douglas Powell advised a patient of mine to lie on a couch two or three times a day, and to lean over first on one side and then on the other, the head thrown somewhat back and nearly reaching the ground, whilst the uppermost arm is extended behind it and the lower beneath the body. In this way the upper half of the chest tends to be distended and the lower contracted, so that the bronchial tubes of the former would be as patent as possible, whilst the larynx, being the lowest point, and the base of the lung the highest, gravity would induce the secretions to flow out of the mouth.

The Pneumatic Cabinet is a mode of producing

pulmonary calisthenics which is perhaps more useful in the prevention than cure of cavities—*e.g.*, in re-expanding collapsed lung, &c.\*

I feel convinced that the difficulty of getting patients to carry out any calisthenic proceedings with perseverance has prevented these obtaining the high therapeutic position they would otherwise have held.

(2) **Exhibition of Drugs by the Mouth.**—The number of these is legionous and I shall best consult your advantage by confining myself to one of which I have had fair trial. I mean *iodoform*. This is a drug containing 96 per cent. of nascent iodine : it is not perceptibly soluble in water, but is so in fixed or volatile oils ; it is therefore best exhibited in pills, capsules, or cod-liver oil, or it may be dissolved in oil of eucalyptus (which is itself of very high value in pulmonary complaints) and a few drops taken on sugar. It is very unirritating and is very slowly absorbed from the stomach, traces being found in the urine three days after the ingestion of a dose. Its absorption is probably aided by the fat in the food (Brunton). Its utility in lung disease was first pointed out by Semmola in 1878; he had used it in cases of bronchial catarrh, asthma, and bronchopneumonia; he also found that cases of incipient phthisis were cured by it, and that in even advanced cases expectoration

\* For details regarding this cabinet and the principle of pneumatic differentiation, see a report by myself in *The Birm. Med. Rev.* for October 1887, and also Dr. Theodore Williams in his Report to the Medical Society of London in the *Brit. Med. Jour.* of May 4, 1889.



is lessened, cough and fever abated, caseation arrested, and life prolonged.

Dreschfeld used it in a large number of cases and found increase of weight and appetite, with lessening of temperature, night-sweats, cough, and expectoration.

Shingleton Smith in 1884 gave an analysis of 46 cases, showing that in 29 there was an absolute increase of weight under its use, and that in the remaining 17 the loss of weight was small, and in many of these the wasting, which had been rapid, was more or less completely arrested.

In a private patient with bronchiectasis I gave it with a double quantity of tar—gr. j. iodof. and gr. ij. tar—to the pill. He began with six pills daily, and increased these in two months to twenty-two, at the end of which time all his moist sounds and rhonchi had vanished, his cough was much less troublesome, and he had gained three pounds in weight.

The man with pulmonary hydatids in both lungs (p. 263), also took iodoform for months at a time. In his case I pushed the drug to 30 gr. per diem, with doubtful result on his symptoms. I may say here that Lazansky gave as much as 90 gr. a day in syphilis (Wood); but Shingleton Smith considers 30 gr. the safe limit, whereas he considers that to render the whole body antiseptic as much as 40 gr. must be exhibited.

But here we must remember the slow excretion of the drug; indeed, M. Mracek in 1882 injected 85 gr. in thirteen days, and found that during these thirteen

days only 20 gr. were excreted, leaving 65 gr. still in the body. It is thus easy, by giving 30 gr. daily, to keep 40 gr. or even 60 gr. constantly in the body. But I feel convinced that as much as 40 gr. is not necessary. Dr. Shingleton Smith's calculation is based upon the supposition that a human being is a mass of inert flesh, weighing so many pounds, and requiring so much per pound of a germicide to keep it from putrefying; but he forgets the *vis naturæ*. "Now by my faith," says Dr. Filleau, "we are no mere culture bouillons." Diseased Nature only requires *assistance*. She is like an over-burdened horse striving with his load up a steep hillside. But, though the horse should come to a weary standstill, yet will his owner not forget his strength, and will seek far less help than if the horse were dead.

(3) **Exhibition of Drugs by the Trachea; that is, Inhalations.\***—Besides having a calmative effect on the larynx, trachea and large bronchi, I must say I have never seen much benefit arise from this method of medication. I have never, for example, seen the fœtor of bronchiectasis remedied by them. I admit that opinions as to their value are greatly at variance; but this very variance is sufficient to make one hold aloof from a method of treatment which is both laborious and unpleasant to the patient, and must indirectly do him harm by limiting his respiration. If they are

\* The intra-laryngeal injection of drugs into the trachea is now obtaining considerable advocacy, and is, I think, to be preferred to inhalations in the treatment of pulmonary lesions. (1895.)

employed, a solution of iodof., gr. 20 ; ol. eucal., m20 ; rect. spirit, ʒss ; ether, ʒss, will be found very serviceable. It should be used on a sponge in a naso-oral inhaler twice a day for two hours at a time. In chronic cases inhalations of medicated steam should be avoided, on account of the relaxing action of the water vapour on the throat and trachea making these very troublesomely liable to catarrh. To the inhalation of cold pulverised fluids this objection does not apply.

(4) **Subcutaneous Injection of Drugs.**—Clinically this has many disadvantages as compared with administration by the mouth, and theoretically it has no advantage, and must be administered in proportionately equal quantities. For an acute effect it is, of course, much to be preferred ; but in these cases we prefer to gradually get our patient under the full influence of the drug, and so avoid the danger of any unpleasant symptoms. There does not therefore at present seem any reason for making use of this method.

(5) **Intrapulmonary Injections.**—This is a method of treatment which holds out considerable hope for the future. There are already a great number of cases recorded in which it has been tried (Pepper, Beverley Robinson, Ransom, &c.) ; naturally most frequently in tuberculosis. That with care it is a comparatively harmless procedure seems well proven. Professor Riva, of Pavia, made upwards of one hundred injections of 40 to 50 cc. each, of a 1 in 3000 solution of mercurial

bichloride, with no ill result. Dr. Shingleton Smith, of Bristol, has extensively used various solutions. He first tried iodoform, and preferred it dissolved in absolute ether, in which it is soluble in the proportion of 1 in 8. This had "only the disadvantage that, by causing temporary giddiness and head disturbance, the patient becomes timid of the injection and unwilling to have it repeated." All other solutions of iodoform or iodol he found acted as local irritants, and could not be "safely or harmlessly" used. The amount of iodoform used in each injection was about one grain. Camphor carbolate was next tried. This is the clear fluid obtained by dissolving three times its weight of camphor in a 95 per cent. solution of carbolic acid. This solution was giving excellent results in injections of 30 to 60 minims, when on the sixth administration to a case of advanced phthisis, the fluid entered a cavity and thence flowed into a bronchus, producing acute suppurative bronchitis with death in forty hours, showing that though innocuous to connective tissues, its action on mucous membranes was far otherwise.

Biniiodide of mercury was then resorted to, the following solution being employed :—Hydrargyri biniiodidi, gr.j; potassii iodidi, gr.j; sodæ phosphatis, gr.ij; aq. destillatæ, ℥50, as much as two-fifths of a grain of the mercury salt being injected at once. This gave some very encouraging results, and no ill effects occurred. This drug, indeed, seemed to Dr. Smith the most suitable of all ; though Villemin states that its

antiseptic properties as regards the bacillus of tubercle are very feeble indeed.

During a considerable experience of intrapulmonic injection with various drugs Dr. Smith encountered the following mishaps—

- (1) The acute suppurative bronchitis above referred to.
- (2) Acute pleuritis : two cases with speedy recovery.
- (3) Pneumothorax, temporary and harmless.
- (4) Intense pleuritic pain, lasting hours, and requiring full doses of morphia.
- (5) Violent fits of coughing.

With the exception of the first, none of these are of sufficient moment to offer a bar to the practice of this treatment, and I believe it has a considerable future before it. The different action of a drug upon the lung parenchyma and the mucous membrane of the bronchus is certainly of great importance, and before using indiscriminately any special solution, the nature of this double action should be carefully investigated by experiments on animals, in the first instance, and afterwards at the bedside.\*

(6) **Incision.**—In the single cavity, acute or chronic, incision seems to be just as much a necessity as in acute empyema ; even in pyæmic cases I think this would be the correct treatment. Localisation is the great diffi-

\* The above remarks refer only to those cases where the lesion is strictly local, and the cavities very few in number : one cannot, in this manner, inject a sufficiency of the drug to combat constitutional disease. (1895.)



culty to be faced. This is no doubt often impossible, but much can be done by the way of circumstantial evidence in making a very shrewd guess as to the position. No straw of evidence should be neglected; the key is often found rather in a wise estimation of the symptoms than in a minute physical examination. Naturally, exploring with a needle which will easily carry pus should be first undertaken, and the exploration should be thorough. Griffith, in his excellent article on empyema (*Medical Chronicle*, March 1889), has some cogent remarks on the necessity of thoroughness. If, he says, in a case of empyema "when from the symptoms we have a reasonable suspicion that pus exists, we should *go on till we find it*, anæsthetising the patient if necessary, and proceeding systematically. In one case it was not until nine punctures had been made that pus was found, and why we did not get it before I do not know; and in another case a double empyema was discovered after repeated exploration."

With these remarks I thoroughly agree; indeed I believe that in this as in many other instances in medicine, much is often lost from mere lack of energetic perseverance on our part. We lack a sufficiency of Dr. Moxon's "initial" energy, so that the biassed and unwise resistance of the patient baffles our weak determination. The exploring syringe should be capable of holding not less than a drachm, and the hollow needle should at any rate be not less than four inches long. If after several punctures no pus be found, this needle should be replaced by a six-inch one. Whenever blood

appears in the syringe this should always be slowly and carefully filled ; by this means the lung may be artificially leeches to the extent of an ounce or more. To those who have often gazed on the gorged tissue around a lung abscess, or have seen the still more tensely filled lung in which exudation has gone too far for resolution, and which would have become an abscess had death allowed it, the great advantage gained by this procedure will not be underestimated, especially if they have examined the dilated capillaries and the microscopic alveolar and interstitial hæmorrhages. In fact, I feel sure that if we could always reckon on withdrawing blood by the exploring syringe, "exploration" would be a desirable method of treatment for its own sake quite apart from its diagnostic value. Unfortunately, as a rule, this does not take place. Such withdrawal should be made gradually, so as merely to relieve the local tension and not attract blood thither from other regions. By acting thus, any tendency the puncture may have given to cause hæmorrhage will be obviated.

The surgeon's great bugbear in this matter of incising lung appears to be the fear lest opening the pleural cavity may cause collapse of the healthy portion of the lung, with its consequent danger of fatal fainting. Another great objection is the fear that a suppurative and general pleurisy should be set up. By many it seems to me that both these objections are made far too much of, for these reasons :—(1) The first is equally present in opening any acute empyema ; yet we do not hesitate to do this, and any serious mischief resulting

from this cause is of extreme rarity. (2) If any portion of a lung be so diseased as to form an abscess cavity, there is pretty certain to be at least one-half of the whole lung *hors de combat* and incapable of collapse or being of much present value to the patient. (3) Dr. Samuel West has shown (*Lancet*, vol. ii. 1887, p. 353) that the force of cohesion of two smooth membranes, such as the pleuræ, is almost double that of the elasticity of the lung (12·5 mm. Hg. as opposed to 7). It is evident, therefore, that if air be admitted into a portion of the pleural sac, there is no reason why it should separate the pleuræ through the whole of their extent, provided they be healthy. This is doubtless the explanation of those small local cases of pneumothorax which occur in apparently healthy people, or after pulmonary injection.

The incision once made and the cavity emptied, the question arises of its further treatment. The course usually adopted is to use a single, large-sized drainage tube. The cavity is then either washed out once a day with some antiseptic solution, or left alone so long as free drainage appears to exist. But of late some have preferred to treat empyemas by perflation.\* And I see no reason why the same method should not be advantageously applied to lung cavities. Less force, however,

\* Perflation—"through blast" (Wm. Ewart, *Lancet*, vol. ii. 1888, p. 226).—In empyema it has the following objects:—(1) Avoidance of all fluid injections and their irritation. (2) *Complete* evacuation of pleura (this seldom done by fluid injections).

Apparatus.—Bottle 8 inches high with  $1\frac{1}{4}$  ins. sol. carbolic ac., 1 in 10. Cork with two tubes, one dipping into sol., one just

would have to be used, for the lining of a pulmonary abscess is usually friable and easily broken down. A strong blast would thus penetrate into the lung parenchyma, and might cause serious and destructive emphysema. If the track of the incision be a dependent one in the usual position of the patient, the simple, unaided drainage tube is probably quite sufficient.

(7) **Resection.**—But, unfortunately for our patients, the most trying cases of lung cavities, and those where interference seems most to be desired, are those in which the cavities are not single but multiple, for the most part small and sinuous, and imperfectly separated from each other, the whole forming a tortuous labyrinth whose communications are diseased bronchi. The walls of this labyrinth are composed of very vascular and unyielding fibrous trabeculæ, which persist in keeping the cavities open and forbid any attempt at their collapse. Hence, the expiratory efforts of the patient for the most part vainly endeavour to squeeze out the secretion contained in them. The labyrinth is a pus-logged one, and withal exposed to the decomposing effects of ordinary air. The pent-up pus naturally becomes thin, serous, and extremely foetid, and is liable to set up a form of chronic septicæmia.

piercing cork. Richardson's bellows and india-rubber tubing. Tube may be inserted several inches, and one hole is enough.

Advantages.—(1) Air diffuses into every nook and cranny. Hence (2) influences every corner—fluid stream only spurts on one or two points. (3) Water merely dilutes fluids and suspends light solids, but either of these sinks in air; hence if aperture be at *lowest point* all these will be driven there.

We have, then, two attempts to make—to establish free drainage, and to induce the trabecular walls to take on a healthy, granulating action. No doubt this latter would follow if free drainage could be established ; but this, I am afraid, is an impossibility. The cavities are not counted by ones, but by tens. And I think the most ardent surgeon would hesitate before he stuck the base of a chest with drainage tubes till it resembled the fretful porcupine. Yet this is what a well-known London physician seriously told me he would have done to himself did he possess such a lung. Theoretically such free drainage is no doubt the correct treatment ; let us hope that soon it will also be that which is found to act best in daily practice.

But we must not forget that, as Dr. Percy Kidd has pointed out, it is the trabeculæ which cause all the difficulty. Simple drainage would hardly be sufficient ; the lung would then resemble a dropping stalactite cavern. It is these stalactites, this trabecular scaffolding, which it is so essential to clear away. They are too resistant to fall together and cohere, even after the freest resection of ribs. If coherence partially took place, their structure is so depraved that no reliance could be placed upon its permanency. Sooner or later disintegration would certainly occur. It is for such cases as these that resection of lung seems the only treatment. These fibrous trabeculæ are intensely vascular, and its veins are almost as dangerous as its arteries. Hence the knife can be but little used, and the *écraseur* is to be preferred. Such an operation in a



debilitated subject is not one, I need hardly say, to be lightly undertaken. On the other hand, the condition of the patient is sometimes sufficiently painful, both to himself and his friends, to justify its performance, especially when we remember that the outlook otherwise is hopeless.

## VI. THE CURABILITY AND PROGNOSIS OF PHTHISIS

IN this short communication I wish to direct your attention to one or two points concerning the prognosis of Pulmonary Tuberculosis. With this purpose in mind I have divided all cases of the disease into two great classes—Curable and Incurable.

Different writers have varying views as to the connotation of these terms. For instance, Dr. Theodore Williams, editing the second edition of his father's book, includes under the heading "cured" only those who have lost all the signs as well as the symptoms of disease. But in doing so I think he has been too exclusive in his application of the word, and this afternoon I shall use the phrase Cured Phthisis for all those cases in which there has been an entire absence of symptoms for a period of not less than one year—that is, for those cases where the patient has been able to follow the ordinary avocations of life for this period without let or hindrance.

I do not think any one need quarrel with this use of, perhaps, the best abused word in the medical vocabulary. Phthisis without any symptoms, no deterioration of

health or strength in its victims, is, at any rate, arrested phthisis. And, once arrested, I can see no reason why it should not always remain so, if the subject of it but continue in the way of life which he pursued during the year of arrest. And, indeed, when a recrudescence of the disease arises it is almost universally traceable to overstrain or excessive exposure.

I do not wish to maintain that a patient who has once suffered from phthisis is not more liable to a fresh attack than one who has not so suffered ; doubtless his liability to it is increased : but cannot we also say the same regarding bronchitis ?—yet we speak of patients being cured of this disease when at any time they lose all symptoms of it.

Phthisis is scarcely a disease in which we can expect all the *signs* of its ravages to vanish : it is hard to believe that some portion of lung can be solidified, softened, and excavated, and yet leave behind no flattening, no impaired movement, no dulness, nor any small change in the character of the breath sound to tell the tale of its previous existence. Yet even this extreme test was satisfied by  $3\frac{1}{2}$  per cent. of 1000 cases who had been under Dr. Williams's treatment for a year and upwards. But, if we take the easier test which I have elected to choose to-day, the percentage at once rises to  $28\frac{1}{2}$ . Allow me to read to you from Dr. Williams's book an abstract of a typical example of this class of case.

A young gentleman, aged 15. May 11, 1857 : Maternal aunt died of phthisis. In last Christmas holidays, after a chill,

had pain in right side, and some cough ever since. Ten weeks ago had a severe chill after playing at football, and since has suffered much from cough and weakness, loss of appetite and breath. Dulness, defective motion, and breath sound in whole right chest. Tubular sounds at and above scapula.

June 16, 1857: Much better. Cough nearly gone. Says breath is not short now.

November 22, 1866: Much improved in general health and strength, but has always found himself rather short-winded on exertion, and sometimes rather wheezy. Still, has kept his terms regularly at Cambridge, and was Senior Wrangler last winter. Six weeks ago he caught a cold, and has had slight cough since, with an occasional feeling of faintness. Bronchophony above right scapula; percussion and breath sounds generally clear.

1868: Continues well.

May 3, 1871: Has continued well generally, but had cough several times last winter. None lately. Is pale and out of condition. Chest sounds good generally, but still tubular sounds in upper right.

1887: Alive and well, and actively engaged in scientific pursuits, experimenting, writing, and lecturing.

I appeal to your sense of fairness and to your experience of the way in which we speak of the healing of disease in general, and I ask you whether, if such a history had been told you of any other disease whatever which has a place in medical nomenclature, whether you would not have said that this lad had been cured of his disease.

Amongst the fairly well-to-do classes then we may reckon upon  $28\frac{1}{2}$  per cent. recovering from an attack of phthisis.

But even this I consider far too rigid a method of estimation, for many of the first attacks of phthisis are so slight as not to come under the care of a medical man at all, and many others that do come under our ken are not diagnosed by us, being classed as colds (from neglect of examination), or bronchial catarrhs, or pleurisies, or catarrhal pneumonias. Dr. Harris, of Manchester, found evidence of previous pulmonary tubercles in 54 out of 139 cadavers of persons over twenty, in which the cause of death was stated to be other than phthisis; *i.e.*, in 38·84 per cent. of people who died from causes other than phthisis evidence of healed phthisis existed.

Now, one-fifth at least of all people who die above twenty die of phthisis (Lombard, Ransome). Hence the 139 deaths represent only four-fifths of the total number of deaths, this total number thus being 174.

Therefore, of any 174 deaths above twenty, 35 (one-fifth) will have succumbed to phthisis, and 54 of the others will give evidence of having recovered from phthisis. From these figures we may deduce the conclusion that of every 100 persons attacked by phthisis, 60 will recover and 40 succumb. All deductions from statistics must be taken *cum grano salis magno*, and I would not have thought it worth while to have placed these before you, did they not agree with my own previous conviction based on my own experience and borne out by analysis of the cases recorded in my private notebooks. Indeed, I am of opinion that these figures rather understate the case,



and that two out of every three persons attacked by consumption recover from that attack, and remain in thorough health and strength with no sign of any local activity in the disease for at least a twelvemonth.

The actual percentages of my own cases are these—

Number of deaths, 16·5.

Number of recoveries for a year or more, 25.

Number still suffering, 32·5.

Not known, 26.

We must remember too that phthisis is one of those diseases which is on the wane. The percentage of deaths from it is rapidly diminishing. It has ever been in the history of disease notable that, when the *extent* of the ravages of any malady lessens, its virulence also lessens—*i.e.*, the ratio of the recoveries to the deaths of those attacked is a constantly increasing one. In other words, the malady becomes more curable. This is especially the case with phthisis, which is essentially a disease of evil hygiene.\* In regard to this disease we are therefore in the happy position of being able as the days go on to hold out brighter and brighter prospects of recovery—of being able ever to give a more favourable prognosis than the collected authority of the day may warrant. To me, around a phthisic the halo of hope cannot wane, however gloomy the immediate atmosphere may show: never do I feel justified in giving way to despair—the beautiful wanhope of our forefathers.

\* Nothing could prove this more conclusively than the great decrease of its prevalence amongst the healthy occupants of barracks following upon improved ventilation and drainage.

How are we to tell what cases are thus capable of cure? The question is an extremely difficult one, and it will be easier, I think, to define it in a negative way by stating all those classes of consumption *not* so capable. Roughly these are four :

(1) **Acute Tuberculosis**—that is, a rapid deposit of miliary tubercle in both lungs, and probably in other organs, producing severe continued fever; great dyspnoea steadily increasing; extreme weakness and a dry cough; the breath sounds everywhere masked by rhonchi and fine crepitations, while the percussion note has an emphysematous resonance. The duration of such cases is two to six weeks.

(2) **Acute Pneumonic Phthisis**.—This usually begins with many of the appearances of pneumonia, but usually, as soon as any physical signs are detected, both lungs are found involved, and there are coarse crepitations scattered in various places throughout them. But more characteristic than these are the grave constitutional symptoms of extreme weakness, intense sweats, and rapid wasting. Excavation speedily ensues and death results in two to six months. If there be no break in the symptoms death usually comes at the earlier date, but sometimes there is a deceptive lull—the temperature falls, the appetite returns, some strength may come and flesh; but soon a recrudescence arises and carries off the patient within the six months.

(3) **Chronic Phthisis with Signs of Activity scattered more or less throughout both Lungs**.—Here the mere local destruction is too great to admit

of a return to health : the patient lies on the brink of the precipice of death over which any slight disturbance may cast him. If there be signs of progressive disease the prognosis should be limited to months, but if the lungs be fairly quiescent, as evidenced by their dryness, with absence of pyrexia and emaciation, he may remain in a state of chronic invalidism of indefinite duration, a duration depending upon his social surroundings and the amount of care he bestows upon his health. At the same time, where there is so little margin between life and death, it would require superhuman precaution and strength of will not to overstep it, and, as a matter of fact, these precarious lives are constantly creating fresh anxiety by fresh lapses of the disease into activity, in one of which the patient is cut off. Probably very few of the cases which have reached this stage live five years, and the majority not more than two.

(4) **Laryngeal Phthisis.**—Once fairly developed this is one of the least hopeful forms. But the only way to make your diagnosis is by a careful laryngoscopic examination, for varying aphonia and laryngeal pain are by no means uncommon in the early stages of phthisis from anæmia or simple catarrh. If, however, you can satisfy yourself of the existence of a caseous tubercle or ulcer, especially if it be at the outer angles of the arytenoids or the interarytenoid mucous membrane, you are warranted in limiting that patient's existence to months, at the outside not more than twelve. As a rule, death is due to the larynx, from the wearing cough, pain and dysphagia it causes. But this is not

always so. Since writing the preceding, a patient has died more than two years after I first saw her with Dr. Hollinshead, at which time very chronic disease in both lungs and secondary, but well marked, laryngeal tubercle were noted, the symptoms of the laryngeal affection having been in existence more than six months at the time of the first consultation. This case is only in accordance with the more recent reports, and I have no doubt, as our everyday knowledge of the larynx improves, so its diseases will appear of less magnitude—will take upon them the real clear-cut outlines of daylight in exchange for their present twilight largeness, for things seen through a glass darkly seem ever rounded with awesome size.

I have made my division into curable and incurable cases, upon the basis of pathology. For I have never seen, nor have I ever come across, a record of a post-mortem where the lesions arising in these four classes were disclosed, in which they were not the actual cause of death. On the other hand, I have either myself seen, or have read reliable records of, post-mortems containing all the various lesions of the curable cases, about to be described, where they were not the cause of death, but in which death arose from some quite other disease or accident.

By the courtesy of Dr. Suckling I am enabled to show you one such specimen. It is a lung of a man who died of acute lobar pneumonia, a disease which you are aware is no more fatal in phthisical subjects than in others. You will see that the whole organ is in a state

between red and grey hepatisation ; but at the apex is a fibrotic depression leading to what on section is seen to be a small sphere of dense pigmented fibroid concentric circles. The intensity of the pigmentation shows that the material of which it was originally composed must have occupied a far larger area in the lung. In other words, there must have been a large excavation which has contracted and solidified, and in doing so has concentrated the pigment. You will also see between the concentric layers small yellow masses of old tubercle which, being extravascular, are unpigmented. This specimen, I think you will admit, is a most striking instance of healed phthisis. It shows well how much disintegration of the fibroid material must take place in order to set free any caseous matter, and so allow of auto-infection.

Let us now then leave the Castle of Despair and enter upon the Delectable Mountain of Hope.

1. *The first class consists of those cases where there are lesions scattered throughout both lungs, but their deposit has taken place slowly and not simultaneously, so that some have fibrosed before others arose or whilst these others were forming.*

The degree of health and strength will depend upon the total amount of lung put *hors de combat*. If this be great and the patient's labour physical, then the strain on the heart will be great, and dyspnœa with palpitation chronically complained of.

Such is the state of the young man whom I now show you. He is a machine tool-maker, and the strain of his



toil is too severe for his enfeebled frame, so that he has to rest for a week or so every now and then. He was treated by Mr. Marsh for a scrofulous ankle, and when that was cured Mr. Marsh handed him over to me. He gives up because of dyspnœa, palpitation, and a hacking cough, and, if his lungs are examined at these times, some dry creakings are discovered in them ; but on closer examination these are found to evidently depend upon heart failure, and are not fresh inroads of tuberculosis, for the heart is large, distorted on its axis, and tumultuous in its action, unmistakably overstrained. And, moreover, remedies applied to strengthen the heart soon dry the lungs and relieve the cough. Still his outlook is hopeful : he is 21 ; through several years of growth he has withstood this strain upon his economy ; and now, every succeeding year will make his burden easier, and at the same time give him more strength to bear it.

The result is far from being so good as it might have been. And why ?

1. Because he was unable to rest whilst the disease in his lungs was progressing.

2. And far more importantly, he was unable to rest whilst the healing contractions were altering the anatomical relationships of the chest cavities, and were in particular dragging his heart from its accustomed bed and depriving it of the support of that resilient cushion which healthy lungs afford it.

3. He is not of strong build : seven out of thirteen of his brothers and sisters died in infancy : his work

under the best of circumstances would leave him little margin : with the respiratory power of his lungs greatly weakened and, chiefly, with the potential force of his heart so sadly impaired he can have no margin at all, so that a passing catarrh or digestive disturbance may suffice to lay him aside for a time.

But if he had been able to rest during the whole period in which the internal anatomy of his chest was re-arranging itself, then his heart would not have suffered ; all his organs and tissues would have accommodated themselves to his lessened respiratory power, and he would have taken up his work on a somewhat lower level of strength it is true, but his whole body would have accurately adjusted itself to this level ; and when his work was too much he would have grown tired altogether, and, as a necessary consequence, must have lessened his labour, and so have safety-valved his life.

There is nothing more dangerous than one, or even two, weak organs in a well developed body. The strength of the chain of life can only be truly measured by that of its weakest link ; but if this be hidden within the trunk, how is its unfortunate possessor to estimate its strength. In the struggle of life the incentive to work is ever before him : his healthy organs urge him to his task, for to them its accomplishment is but pleasure, and so on he labours till his wretched weakling cries aloud with pain and utter undoneness, and forces him to cease his toiling.

Surely such cases as these are living witnesses to the

need of charitable succour for the consumptive : as yet the phthisical poor are the pariahs of medical charity. Into our ordinary hospitals they are only admitted *sub rosa*, or for the treatment of some intercurrent ailments. A sloughing chancre or gonorrhœal knee is admitted without question, but to the phthisical our lips are set with sternness and the wards of our hospitals are closed, unless, indeed, the victim belongs to the great class of pauperdom : then we go to the other extreme, and allow him to luxuriate in a palatial mansion under the skill of one of our ablest physicians, till such time as it may take him to get well of his phthisis : but to the self-respecting poor opens out no such Paradise.

2. *The second class of curable cases is that where both upper lobes are involved, but the lower present no signs.*

This woman whom I now ask to come forward is an instance of this kind. Since she had congestion of the lungs at the age of twelve she has always been ailing, but chiefly with dyspeptic symptoms. She has now the signs of consolidation down to the fifth rib on the right, and those of fibroid cavities throughout the upper lobe of the left lung in front, with also the signs of consolidation at the left base. But during the last six months, whilst she has been under my care, she has had no active mischief detectable in the lungs, nor any symptoms referable to such. Were it not that she has to look after her children and see to her house under bad hygienic conditions, I have little doubt that she would soon get strong and healthy.

Another favourable example of this class is a young

University man, fellow of his College, who was athletic and in good health up to 1890, when, at the age of 22, he became phthisical, spent two winters at Davos under my friend Buol, where he submitted to a thorough course of Koch's treatment, and then in 1892 came to ask me whether he were fit to take up his duties as College lecturer. Both upper lobes were widely affected, there was bronchial breathing and dulness, with dry crepitations. But constitutionally he felt well, and I advised him to run the risk of returning, as much depended on it. He did so, and up to the present, Sept. 1894, he has kept quite well and strong, acting as bursar and lecturer in his College.

3. *The third class, where the mischief is confined to one lung, gives us still more hope.*—For though the mischief may be large in amount yet its distribution is limited; it is more compact and presents fewer weak points where the protecting fibrosis may give way and set free the enclosed tuberculous material.

Perhaps the best illustration I can give you of this condition is that of a sallow, thin, dark-haired woman of 30, whom I had under my care for six months when house physician at Brompton. On admission she had signs of softening over most of the right upper lobe, with some crepitations in the lowest: she was much prostrated, hectic  $96^{\circ}$  to  $104^{\circ}$ , heavy night sweats, and, worst of all, anorexia and vomiting, with irritative diarrhœa. So bad was she that for three months it was with her a matter of life and death: meanwhile a large cavity formed, and at the end of the fourth month it

began to contract. All the symptoms rapidly ameliorated, and two months later she appeared well and left the hospital to resume her work : six months later, when I lost sight of her, she still remained well.

4. *Lastly, the fourth class is full of hope. It consists of those cases where an attack of phthisis has remained limited to one upper lobe.*—It is very seldom, under these circumstances, that we have to take a gloomy view of the matter, provided the patient is able to lead a fairly healthy life.

One of the hardest working clergymen in this city, a tall muscular man of 53, came to me in April 1890, complaining of feeling ill and out of sorts, with a cough and pain in his right side, these symptoms having continued for six weeks. He told me his chest was weak, and wished me to examine him first, and that then he would tell me his history. I did so, and found all the signs of a cavernous condition on the right side anteriorly down to the fifth cartilage and also in the upper axilla : he also had wheezy crepitations throughout the right lung.

He then told me that fourteen years ago he had a bad attack of consumption and was given up by the physician who saw him in consultation : getting better, he went to see Dr. Theodore Williams in London, who told him he then had a cavity at his right apex. He wintered two years at Cannes and Hyères : has had coughs and colds several times since, but, with the aid of his month's annual holiday, has always been able to keep to his work.



And this work is no sinecure : he is a High Church-man, and so has many services, and also visits the houses of the poor from seven to ten most nights. He has his morning cold tub, and dries himself by making *sixty* movements with his dumb-bells.

On further examination I found that his heart was overworked, that his arteries were thickened and somewhat tortuous, and that his pulse was 100 and too hard and sudden. He also had lost his appetite, and was fastidious and languid.

It was evident that his heart was the organ chiefly at fault : it had three difficulties to contend against—the forty days fasting through Lent, degenerated arteries and a damaged lung. With a cardiac and stomachic tonic and partial rest he soon picked up, took his annual holiday earlier, and returned in his usual health. [1894. He still remains well.\*]

You will have perceived that I have really confined myself to one point in prognosis. I have supposed a medical man called in to see a patient suffering from phthisis in an active form, and that the anxious relatives have asked him the question, Will he get well ?

\* I have since seen his elder brother who was suffering from left pleurisy and a broken-down enlarged heart with degenerated arteries. In him I found undoubted evidence of a fibrosed cavernous state at the left apex, and learned that, twenty years previously, he had been laid up for three months with lung mischief. The constitutional tendency to fibroid change stood these two brothers in good stead as an extinguisher of the tubercular inflammation. It is because fibroid degeneration shows itself chiefly in the vascular system that the theory, otherwise untenable, has arisen of the antagonism of cardiac disease and tubercle.

I have indicated those conditions which, in my opinion, make it our duty to say No uncompromisingly. I have indicated those conditions which, in my opinion, justify us in saying that recovery is possible, and moreover, in the last two classes, I have tried to show that we are justified in using the term probable instead of possible.\*

After all, this is the momentous question in phthisis as it is in all active forms of disease. We are not asked whether life will last 5, 10, 15, or 20 years should recovery take place; but, rather, will this recovery occur at all? And it seems to me, that this is rightly the essential question. Equilibrium once restored, its stability is a secondary matter: it is by no means the patient with the greatest stability whose life must be longest; long life will rather be accorded to him who, knowing the amount of his stability, is careful never to step beyond the bounds of this, whether it be large or small.

\* Further experience would make me more hopefully modify these dicta from No, Possible, Probable, to Very little chance, Chances equal, Chance greatly in his favour. But I must not omit to say that in this paper I have not touched on conditions other than anatomical, though these—*e.g.*, Age, Heredity, Social Surroundings, Pecuniary Condition, Occupation—are often of even greater prognostic importance than the ones I have considered. (1895.)

## VII. THE INGLEBY LECTURES ON THE CONDITION OF THE VASCULAR SYSTEM IN ANÆMIC DEBILITY

### LECTURE I. THE BLOOD—ŒDEMA

MR. DEAN AND GENTLEMEN,—It is fitting that I should first speak in pious remembrance of Dr. John Tomlinson Ingleby, the great obstetric physician, who served this College so nobly, and in whose honour we are met here to-day. The tale of his life is briefly this: Born at Cheadle in Staffordshire on March 7, 1794—one year after the death of John Hunter—he had a good preliminary education; for five years thereafter was apprenticed to Mr. Bourne, of the same town, and at the end of this period pursued his studies in London and Edinburgh.

In May 1816, he settled in Birmingham as a general practitioner, and in August of that year was appointed surgeon to the Dispensary, in succession to Mr. Grainger. When, through the astonishing zeal of Mr. Sands Cox, this school was first formed in 1828, Mr. Ingleby was chosen to lecture on Midwifery; and when, in 1834, through the public spirit of Mr. Sands Cox and Dr. James Johnstone, our school was inaugurated afresh in the permanent building we are so soon

to leave, Mr. Ingleby was again elected Lecturer on Midwifery. At this time each lecturer defrayed a proportion of the expenses incurred in the delivery of his course and received no emolument, but in 1836 this variable amount was transmuted to a fixed payment by the lecturer, which, in the case of Mr. Ingleby, was £14 yearly. He lectured at a quarter to eight on the first five mornings of the week, his course consisting of sixty lectures. This post he held till his death ; but in 1838 his increasing practice obliged him to petition for assistance, and in 1839 Dr. Berry was appointed to help him. In 1829 he was chosen surgeon to the Asylum for Magdalens, a position he resigned in 1843, having previously, in 1840, left the Dispensary, of which he was then senior surgeon.

In 1832 he published his masterly treatise on "Uterine Hæmorrhage," which was at once acknowledged to be the most complete and comprehensive essay on the subject in the English tongue. A few years later, 1837, came "Facts and Cases in Obstetric Medicine," dedicated to the students who had attended his lectures since the commencement of the school.\*

These two works obtained for him a European reputation, and through them he was made M.D. of Heidelberg and F.R.C.S. of Edinburgh. Both these volumes, which I show you here, bear unmistakably,

\* I have since discovered several able articles of his in the *Edinburgh Medical and Surgical Journal*, 1835-1840, which have the further interest to us of being admirably illustrated by Mr. Alfred Baker.

even to the superficial reader, the impress of a master. The language approaches that peculiar character which we so greatly love in Trousseau and Watson. It is not eloquent in the usual acceptation of the term ; there is no profuse verbiage nor resounding phrase : but there is something better. Dr. Ingleby's words are well chosen and to the point. His language is concise, and yet not cramped ; it reminds one of the placid leisure of the study arm-chair, where each sentence is lovingly thought out, with the aim, not of thrusting upon you much knowledge, but rather of putting what he has to give you in the best possible dress, and yet you seem ever pervaded with the calm reticence of much learning and keen observation. It is in the relation of cases where he appears to most advantage. These he places before you with such simple directness, such calm unexcited realism, the salient features alone being sketched in, that you seem to be at the bedside of his patient listening to his exposition rather than reading it from a sixty-year old book. In a word, his writings live. Such style as his seems almost a lost art now-a-days. I know not whether it be that the wonderful development of scientific knowledge has gone far ahead of the growth of language, but the writer of to-day is ever pressed down by his wealth of facts, he fails to marshal them into an organic whole, but satisfies himself with their bare repetition, resting well content if he can but contrive to put them all down with no single omission. He forgets that six facts told memorably are worth far more to the world than sixty put down with vague incoherence. For the



six the reader will bless him with keen animation, but the sixty he will mostly skip with weary annoyance.

In Birmingham medicine, Dr. Ingleby made two innovations. From the time he was appointed Lecturer in the School, he devoted himself to the study of Midwifery, and was the first specialist in Obstetric Medicine in this city. Secondly, in the winter of 1840-41, he gave a course of lectures to the practitioners of the district, which we should now designate "Post Graduate." As a lecturer, a contemporary speaks thus of him: "He was neither eloquent nor fluent, but always in earnest, and he treated his subject with the skill of a master; he had no learned speech, but he used good Saxon words with a taste and precision which riveted the attention of his class. In description and detail few could excel him. In all respects he was an accomplished teacher, and not a little of the reputation of Queen's College is due to his occupancy of its Midwifery chair."

As an obstetric physician, he was without a rival in the provinces, and more than one eminent London specialist sent to him patients for his opinion. His fame, though somewhat slow in coming, was very great. During his last years he may be said to have spent the whole of his days amongst his patients. He had many long journeys, and in his rooms saw patients from afar. Though eminently successful in the conduct of his cases, he was almost timid in his diagnosis and treatment. This was mainly due to a very nervous and excitable temperament which he had inherited, a dangerous case

depriving him of both sleep and appetite ; but it was also due in part to his keen sympathy, a sympathy which forced him to take the greatest interest in the social as well as medical condition of his patient.

Dr. Ingleby was of middle height and spare build. He had a peculiar and rather awkward gait, swinging his arms and legs in a strange and most characteristic manner. His eye, though small and restless, would often light up with a rare brightness and intelligence, and his expression was kind and gentle. His character was one of singular amiability and affectionate domesticity. In religion he was an orthodox Churchman, and his piety was fervent and sincere. His professional conduct was straightforward, simple, and free from all arts and devices. In consultation he was unassuming and deferential almost to a fault, and never spoke with discourtesy or disparagement of a fellow practitioner. From first to last he worked at his profession with enthusiasm ; “at any time, by night or day,” says the writer above quoted, “without fee or reward, he was ready to assist whomsoever might chance to require his services.” And finally, in 1845, after vainly struggling for two years against failing health, he dies of atonic gout, in the fifty-second year of his age, a martyr to the art he loved so well.

Dr. Ingleby's only son—Charles Ingleby—entered the Church and died in 1873. He left as a trust to his executors a large sum to be divided amongst various charities ~~and~~ institutions. In August 1876, Mr. George Paulson Wragge, a Birmingham solicitor, the

sole surviving executor, gave, out of this trust, £2000, "to establish in connection with this College, The Ingleby Lectures and Scholarships for promoting the advancement of Obstetric Medicine and Surgery, including the Diseases of Women and Children, being branches of Medical Science in which Dr. John Tomlinson Ingleby, the father of the said testator, Charles Ingleby, acquired considerable reputation."

To this honourable post, thus created, the Council of Queen's College have thought it good to appoint me ; and for this high appreciation of my merit I thank them most heartily. That you will approve of their choice is my earnest hope, but I am conscious that this hope can be realised only by the generous leniency of your judgment ; for the imperfections of the work I am about to submit to you are great and numerous.

A very large proportion of the adult population of England, indeed, in a great city such as this I would say almost one half, have an organisation incapable of living at the high rate of pressure by which the material success of the capital-lacking individual can be alone attained in these days. Some are wise enough to perceive this ; forego their success and retain their modicum of health. Others are not so far-seeing, but fretfully attempt to keep pace with their stronger neighbours ; but the result is a succession of arduous leaps instead of a steady running along life's road, till by degrees the leaps get smaller and smaller, ending finally in an utter breakdown, from which oftentimes there is no complete rallying, and the rest of life is one

of peevish invalidism, or, at best, but one of senile energy.

To most of us, however, it is not given to choose the amount of work we shall perform ; unfortunately, we find ourselves set labour beyond our strength before we are aware of the weakness of that strength. When we discover our weakness we have but Hobson's choice remaining to us, either to fall out of rank altogether and begin life afresh, or else to continue the uphill struggle against high pressure with an organisation fitted for low pressure exertion only. To impress upon us disciples of medicine the manifest unwisdom of this course is no more difficult than it would be to explain to an engineer the folly of giving a low pressure engine high pressure work to perform. But with the laity it is far different ; to women, young people, and uninstructed males I can understand even the mechanical engineer having difficulty in explaining the dangerous madness of using a low pressure engine for high pressure work ; and if so, can we wonder that the best instructed and best balanced laic minds, with the intensity of necessitous desire obscuring their keenness of vision, should fail to be convinced by us, who are the engineers of their frail humanity, that its frailty is incapable of high pressure action ? *Hinc illæ lachrymæ amicorum* over lives prematurely blighted ; over lives broken down and finished when they should but have reached their prime. Hence also the unaccountable perversion to peevish irritability of some temper previously well under its owner's control. Hence the

sudden outbursts of puny anger over the merest of trifles which go so far to embitter and alienate domestic love. Hence the half-mad rushings to excess in tea and alcohol and various other stimulants with which the poor debilitated one still trusts to bolster up his broken strength a little longer. Hence, later on, when the fight is recognised as hopeless, the fatal indulgences in the various nepenthes which help one to forget the misery of chronic invalidism—the consciousness of never being able again to do a good day's work. As with the breadwinner so is it with the housewife, for her duties to her mind appear equally imperious and exacting. So is it too with the mere pleasure-seeker, who often drags a body more fit for bed than dancing to a weary rout.

With children another evil, that of ignorance and fatal unconcern for health, which they consider to be the care of their parents, is perhaps the chief cause of the overstrain, which they often with utter needlessness put upon their constitutions. And this at a time when these constitutions are developing most rapidly, and therefore require much husbanding of energy. Again, we have the too-ready return to work after the exhaustion of a severe illness, such as typhoid or influenza, when nutrition is but feebly reasserting itself, and, occupied as it is with repairing past ravages, is unable to provide pabulum for fresh exertion.

It is to the more certain recognition of the early stages of these anæmic and debilitated states that I wish to speak to you, for *facilis descensus Averni*, and it is



rarely, except in the chlorosis of girls, that your help is sought for the fundamental condition itself in the first instance, but rather for some trivial symptoms springing out of it, a slight gastric upset, or tracheal catarrh.

A few days' rest, with appropriate diet and medicine, sets this trouble right, and you tell your patient he may return to work. He does so, but in two or three days more comes to you worn out and with a subacute recurrence of the previous malady. He is, moreover, greatly depressed, and perchance nourishes against you a secret grudge for having allowed him too soon to return to his labour. There are few positions more humiliating to the medical man than this, that through a well-meant endeavour to get his patient quickly off the sick list he has brought upon him a tedious and wearisome illness.

This happens not seldom, the patient leading the quiet invalid life at home seems well, and the doctor is perhaps too keenly sensitive to the reproach of keeping him too long on his books; or weakly, against his better judgment, gives way to the earnest representation of the necessity for return to work. Seldom, indeed, is the need of a strong will more urgently required by us.

There is nothing more strengthening to the medical will than a complete certainty of diagnosis. How many of us would be able to withstand the piteous pleadings for solid food of the convalescent typhoid were our knowledge of the terrible danger of giving

way wanting in pathological precision, and doubt existed in our minds as to the *post* or *propter* of the sad result of taking such nutriment. Almost, if not quite, similar certainty of diagnosis may be arrived at in cases of anæmia. I admit this certainty cannot be easily learnt from a text-book as is the case with typhoid ; but a few years' careful and persistent clinical work will give it to us, and enable us to speak as dogmatically to the debilitated patient as the young house physician does to the convalescent from typhoid. Indeed, in the case of chlorosis, the diagnosis of the patient's state is almost as simple as in that of typhoid fever ; yet how comparatively seldom do we see chlorotics sternly forbidden to overtax their strength in the various ways that many girls, from emulation, thoughtlessness, and sad ignorance, almost daily do !

With adults and amongst the poor it may be impossible for us to have our wishes as to the conduct of their lives carried out, but with adolescents we can plead no such excuse in the middle and upper ranks of life. Here we can command, and it is our duty to do so with calm decisiveness, so that the hopelessness of appeal or persuasion may be made quite evident. Boys will want to play football, girls tennis, and both will vehemently object to a daily siesta. But we must have firmly made up our minds beforehand on all such points, and remain steadfast and immovable when once our fiat has gone forth.

In the elucidation of this state I have set myself in these lectures merely its diagnosis as regards the vas-

cular system with its appropriate treatment, and even of the vascular system I can refer only to those points which have more especially come under my own observation.

I will first consider the condition of the **Blood** itself. The quality of the plasma depends upon the nature of the nutriment derived from the alimentary canal; either directly by means of the intestinal capillaries, or indirectly through the lacteals. This in its turn is governed partly by the food, but more especially by the capacity of the intestinal mucous membrane to absorb and modify the food. It is this membrane which absorbs peptones, turns them again into albumins, and, as albumins, passes them on to the blood. It is its villi which absorb fats and pass them on to the lacteals, and which change the small quantities of soluble soaps and fatty acids into neutral fats before so handing them on. Nothing, indeed, seems to be taken up by the blood or lymph through the mere physical forces of endosmosis, diffusion and filtration alone: the cell protoplasm of the mucous membrane modifies all varieties of absorption. Even on many true solutions it exercises a selecting power and takes up some (grape sugar, *e.g.*) which diffuse with difficulty, more rapidly than others (as sodic sulphate) which have a far greater diffusing power; while others (some soluble pigments) it refuses to take up at all.

Practically, then, we may say that for the maintenance of a healthy plasma the integrity of the intestinal mucous membrane is absolutely essential. And when

we remember that this same membrane is chiefly instrumental in the due preparation of the food for absorption, the high importance we must place on its functional health stands out still more plainly. For, from the plasma spring all the more complex elements of the body, the red and white corpuscles amongst them, and hence it is the ultimate foundation from which all our tissues are built.

The corpuscles, both red and white, are formed during extra-uterine life, chiefly, if not altogether, in the red marrow of bone. This red marrow is found in the bones of the skull, in those of the trunk, and in the extremities of the long bones. The red and white have independent origin, the antecedents of the red are called erythroblasts, of the white leucoblasts.

This red marrow consists of fat granules imbedded in the meshes of a lymphatic tissue: through it run numerous vessels; the arterioles are fewer and of much smaller calibre than the venules; the circulation in these latter is therefore slow, and hence plenty of time is allowed for the absorption of nutriment from the plasma contained in them. It is in these venules that the red corpuscles are developed; they appear to have but ill-defined walls, so that passage of corpuscles from them into the tissues and *vice versa* is easily made. The fully formed red corpuscles occupy the axial stream of these venules; at their peripheries are erythroblasts containing very little hæmoglobin, but having a large spherical nucleus and a homogeneous, or but slightly granular, protoplasm. Between these erythroblasts and the fully

formed red corpuscles are many gradations of cells, all of which are actively dividing. The leucoblasts differ from the erythroblasts in being altogether without colour, in their central nucleus being small and of variable form, and in possessing many nucleoli. Both erythro- and leucoblasts have amœboid movements, but these are much more active in the leucoblasts. The leucoblasts are outside the vessels, in the marrow parenchyma, and enter the blood-stream by diapedesis.

The parenchyma of the red marrow consists of a delicate lymphatic tissue containing many fat cells in its meshes. In anæmia these fat cells diminish greatly, or even vanish, their places being taken by leucocytes. The venules also dilate, lessening the space occupied by the parenchyma ; in extreme cases this actually ceasing to be visible, the adjacent walls of the venules touching each other. Inside the venules the axial stream of fully formed red corpuscles becomes a mere streak, and the widened vessels are filled with erythroblasts and, outside them, leucoblasts in every stage of division.

In this way the two great changes in the blood, which are characteristic of all varieties of anæmia are brought about. A functional or organic defect in the intestinal mucous membrane diminishes the proportion of albuminates in the plasma ; and, probably as a result of this, a deterioration in the functional activity of the red marrow impoverishes the red corpuscles and thus lowers the oxygenating power of the blood.\*

\* *Vide* also Report of Dr. E. Lloyd Jones, *Brit. Med. Journal*, July 28, 1894.



I think it only fair to say a few words concerning the spleen, which formerly held so high a position as a source of red corpuscles. That it does so form them during intra-uterine life to a very considerable extent is still acknowledged, but this function entirely, or almost entirely, ceases with the birth of the child. Throughout life it helps to produce white corpuscles, and throughout life it, with the gastro-intestinal mucous membrane, is the most active seat under normal circumstances of the destruction of red corpuscles. Yet it does not seem to be one of the great organs of life, since it can be removed with impunity. Sir Spencer Wells' well-known case of splenectomy is perhaps the best evidence of this, as we have so great an authority as Dr. Dreschfield on the after-condition of the blood. Very briefly it is this: Sir Spencer Wells removed a very large spleen from a young married lady of 21. This lady had had ague at the age of 4, when her spleen was noted to be large, and she was afterwards anæmic, but on coming to England two years later she soon became strong and healthy, and married at 18. Early in her first pregnancy her medical attendant noted that her spleen was increased in size, but this was probably no new thing, as the patient had been cognisant of a lump in her left side all her life. After her confinement, the spleen grew far larger, and she became anæmic, but her blood was healthy, except perhaps for a slight increase in the white corpuscles. Suddenly a severe intra-splenic hæmorrhage took place, and the spleen was removed. Recovery was good, no bad symptom appeared, and a year later

Dr. Dreschfield examined her blood, and reported that it was perfectly healthy, except that the hæmoglobin was only 75 to 80 per cent. of its normal amount.\*

The special and diagnostic blood changes which occur in individual varieties of anæmia are best seen by consideration of the following analyses which I have collated from the records of the Queen's Hospital, with the kind permission of my colleagues :

Case 1.—A girl with Chlorosis and ? Gastric Ulcer.

1st Report. H.B.† 28 p. c.	2nd Report. H.B. 45 p. c.
Red Cs. 33 p. c.	Red Cs. 90 p. c.
White Cs. 100 p. c.	White Cs. 100 p. c.
3rd Report. H.B. 65 p. c.	4th Report. H.B. 60 p. c.
Red Cs. 118 p. c.	Red Cs. 96 p. c.
White Cs. 100 p. c.	White Cs. 100 p. c.

Case 2.—A girl with Simple Anæmia and ? Cerebral Tumour.

1st Report. H.B. 30 p. c.	2nd Report. H. B. 50 p. c.
Red Cs. 100 p. c.	Red Cs. 90 p. c.
White Cs. 100 p. c.	White Cs. 100 p. c.

Case 3.—A child with Simple Anæmia after Typhoid.

H.B. 60 p. c.  
Red Cs. 90 p. c.  
White Cs. 100 p. c.

\* Dr. Malins (*Lancet*, Sept. 15, 1894) reports a case where he successfully removed a spleen for twisted pedicle on Jan. 2, 1894. The blood was examined several times during the succeeding month and found practically normal. On March 30th the white had doubled in number. On May 23rd the red corpuscles had sunk to 3,300,000, and the white cells had risen to 50,000 (from 15,000). This points to the gradual development of leukæmia as a result of the operation.

† Stands for Hæmoglobin.

## Case 4.—Simple Anæmia after Childbirth.

1st Report. H.B. 20 p. c.

Red Cs. 38 p. c.

White Cs. 100 p. c.

2nd Report. H.B. 20 p. c.

Red Cs. 54 p. c.

White Cs. 100 p. c.

## Case 5.—A man with Chronic Nephritis, Mitral Disease, and persistent slight Hæmaturia.

1st Report. H.B. 60 p. c.

Red Cs. 94 p. c.

White Cs. 100 p. c.

2nd Report. H.B. 50 p. c.

Red Cs. 90 p. c.

White Cs. 100 p. c.

## Case 6.—Lad with Hæmorrhagic Anæmia from Bladder Tumour.

1st Report. H.B. 30 p. c.

Red Cs. 49.2 p. c.

White Cs. 75 p. c.

\* 2nd Report. H.B. 28 p. c.

Red Cs. 48 p. c.

White Cs. 100 p. c.

\* 3rd Report. H.B. 30 p. c.

Red Cs. 72 p. c.

White Cs. 100 p. c.

## Case 7.—A man with Scurvy.

H.B. 30 p. c.

Red Cs. 84 p. c.

White Cs. 100 p. c.

## Case 8.—A man with Alcoholic Cirrhosis of the Liver and Strumous Glands in the Neck.

H.B. 30 p. c.

Red Cs. 84 p. c.

White Cs. 100 p. c.

## Case 9.—A girl with Pernicious Anæmia ; Death ; slight Enlargement of Spleen ; Pyrexia towards end.

H.B. not estimated.

Red Cs. 17 p. c.

White Cs. 100 p. c.

\* After removal of tumour.

Case 10.—Man with Pernicious Anæmia and Alcoholic Cirrhosis of the Liver ; Spleen not enlarged ; slight Pyrexia ; 98°.5 to 99°.5.

1st Report. H. B. not estimated.	2nd Report. H.B. not estimated.
Red Cs. 45 p. c.	Red Cs. 108 p. c.
White Cs. 100 p. c.	White Cs. 100 p. c.
3rd Report. H.B. 45 p. c.	4th Report. H.B. 39 p. c.
Red Cs. 44 p. c.	Red Cs. 43 p. c.
White Cs. 100 p. c.	White Cs. 100 p. c.
5th Report. H.B. not estimated.	6th Report. H.B. 55 p. c.
Red Cs. 44 p. c.	Red Cs. 60 p. c.
White Cs. 100 p. c.	White Cs. 100 p. c.

The Red Cs. were always variable in shape ; many were megalocytes, some granular, and some had nuclei. This patient went home much improved ; his treatment being chiefly iron and cod-liver oil.

Case 11.—A woman, age 30, with a greatly Enlarged Spleen and slightly Enlarged Liver ; ? due to Syphilis.

H.B. 23 p. c.  
Red Cs. 53 p. c.  
White Cs. 100 p. c.

Case 12.—A woman, age 50, with Bronchitis and Anæmia.

1st Report. H.B. 55 p. c.	2nd Report. H.B. 78 p. c.
Red Cs. 85 p. c.	Red Cs. not estimated.
White Cs. 100 p. c.	White Cs. not estimated.

These cases exemplify very fairly the main changes in the corpuscles which arise in the various forms of anæmia. I much regret that I am unable to furnish you with the results of examinations of the plasma, so that the analyses might have been complete.

All those which we may describe as Simple or Symp-

tomatic anæmias have a striking uniformity. In all, the number of white cells is normal ; in all, the number of red cells and the amount of hæmoglobin is reduced, the latter to a much greater extent than the former ; hence the oxygenating power of each red cell must be lessened as well as the number of these. Moreover, Cases 4 and 6 show that, as improvement takes place, this is seen far more quickly in the number of the red cells than in the amount of hæmoglobin. In other words, it is easier for the system to produce a larger number of imperfect cells than a smaller number of higher oxygenating capacity. This is what we should expect, as it is only an illustration that quality is ever rarer and more difficult of production than quantity. Case 5 shows the same thing reversed, as the blood deteriorates the quality of the red cells decreases far quicker than their number.

Case 10—if we omit Report No. 2, in which there is probably an error—is a fair example of the state of the corpuscles in pernicious anæmia. The white cells remain normal, the red lessen greatly in number ; but the amount of decrease in hæmoglobin does not do more than keep pace with this lessening, in some instances, indeed, it does not even decrease as much as this, and hence the hæmoglobin capacity of each corpuscle is greater than normal ; but, probably, never its oxygenating powers ; for as this case also shows, many of the corpuscles were much increased in size, and therefore the hæmoglobin would exist in them in an abnormally diluted condition ; hence, though the surface



of the cell would be larger, the amount of hæmoglobin which it could expose to oxidising and deoxidising influences would be no greater than normal. Case 10 shows some of the alterations which the red cells undergo in pernicious anæmia—their nucleation, granular protoplasm, and great variations in shape—all of which changes recall to our mind their embryonic state as erythroblasts. Further, Case 9 shows the extreme diminution in the number of the red cells which may occur before death.

This brings us to the consideration of the nature of the difference which exists between pernicious anæmia and all other forms. This difference is usually thus stated: the essential cause in pernicious anæmia is an increased destruction of the red corpuscles, their production remaining normal, or even rising above normal; whereas, in all ordinary anæmias, the essential cause is diminished production. In the one case we should have an extra activity of the blood-destroying organs, in the other a feeble activity of those organs which make the blood. As the blood-makers and blood-destroyers are not thought to be the same organs, this difference is evidently a great and radical one.

But does such a difference actually exist? Does the theory of such fully explain the conditions? Dr. Hunter tells us that in pernicious anæmia there is an excess of iron found in the liver and spleen, hence, he says, these organs\* break up an excessive number of

\* Or rather, that the spleen does so, and sends on the hæmoglobin to the liver.

corpuscles to obtain the excess of hæmoglobin, which produces this excess of iron. But, on the same reasoning, there should *normally* be an accumulation of iron found wherever corpuscles are disintegrated. Let us confine ourselves to the liver. This organ takes the hæmoglobin set free from the red corpuscles, and produces bile pigment with it; but bile pigment has no iron, so that all the iron which was in the hæmoglobin must be left in the liver. It is true, a small proportion finds its way into the bile as phosphate of iron, but this forms only a small fraction of the total obtained from the hæmoglobin. What becomes of the rest, and, moreover, of that which is absorbed from the food and passed on to the liver in the portal circulation? For iron is found in the plasma of the portal vein, but not in that of the hepatic.

It is evident that in health, at any rate, this cannot remain in the liver, or else this organ would in the aged become loaded with iron. Dr. Delépine has suggested that the liver re-forms it into hæmoglobin to produce new red corpuscles, forwarding it, I suppose, to the bone-marrow for this purpose.\* Whatever happens to it, it cannot stay in the liver; this organ must have a means of disposing of it of which we are ignorant. Now should this unknown function of the liver be but

\* For simplicity's sake one speaks of iron only, but iron is found in the liver cells in union with nuclein and a proteid, and it is some such compound, if not the unstable thing hæmoglobin itself, which is sent to the bone marrow, so that this is deficient, not only in iron, but in all the constituents of hæmoglobin.

feebly performed, we should at once have, as a result, an increase of iron remaining in the liver. Also, it is easy to conceive that, as the accumulation occurred, the bile-forming function would excrete more into the bile as phosphate.\* For this, being a procedure of disintegration, would be far easier of performance than the rebuilding up into hæmoglobin. Should, then, this hepatic hæmogenetic function grow feeble, we have an explanation of the excess of iron found in the liver in pernicious anæmia. In all other anæmias we have evidence of feebleness of hæmogenesis; but in them the liver seems to escape. Is it not natural to assume that as the disease takes on a severer type—becomes pernicious—the liver also fails in its hæmogenetic powers?

Dr. Hunter has shown us that the liver undergoes actual organic disease in pernicious anæmia, for he has found the central zone of the lobules to be the subject of fatty degeneration. Further, he has shown that the iron, which in health is only found in the leucocytes of the branches of the portal vein inside the liver, in pernicious anæmia is also found in the cells of the outer portions of the hepatic lobules. This would seem to indicate that the liver took up the iron, and made the attempt to transform it into hæmoglobin, but as the central zone was diseased and unable to assist, some of the iron had to remain in the still functional cells of the outer zone.

\* Dr. Hunter has found an excess of iron in the urine in cases of pernicious anæmia.

If we take this view of the matter, then pernicious anæmia merely becomes a very severe form of anæmia generally, but no distinct disease. It becomes easy to understand how other forms may gradually become pernicious, and how pernicious anæmia itself may recover by gradually passing into milder forms.

You will say, Surely the condition of the blood, the marked difference in the relation of the red corpuscles to the hæmoglobin, and the changes in the corpuscles themselves are diagnostic marks indicating a fundamental divergence. But even this is not so irreconcilable a state as one is apt to imagine. I have at this present time a young girl under my care (Case 1) in whom the red corpuscles are but 33 per cent., whilst the hæmoglobin is as much as 28 per cent.; yet there is no doubt that she is simply a chlorotic, with, it is true, symptoms of gastric ulcer. She shows no evidence of dangerous illness nor extreme feebleness; moreover, she is rapidly improving under Blaud's pills.\* The corpuscles may be most variable in size and shape in pernicious anæmia, but they are by no means normal in these particulars in other anæmias; chlorosis is recognised as having a partiality for microcytes. It is only to be expected that the cells should most depart from their normal type in the severest form of the disease. Indeed, clinically, we are continually finding

\* Since writing the above the further Reports have been obtained. The girl is convalescent, much improved in colour, and with no murmurs, and has been discharged.

transition forms which more and more bridge the gap that still separates pernicious from simple anæmia. Often it is hard to tell the one from the other, and cases which begin with all the signs and symptoms of simple anæmia end fatally. Moreover, if we take a case of the most fatal and another of the mildest type of anæmia, these two certainly do not differ as much as a chronic tuberculous gland disease of the neck does from an acute miliary tuberculosis.

The **œdema** of anæmia is but ill understood ; perhaps the least so of all forms of œdema, none of which are easy to explain. Its possible causes are :  
1. A morbid change in the blood. 2. A morbid change in the capillary endothelium. 3. Deficient action of the heart.

The blood in anæmia is often unduly dilute ; but mere dilution will not cause œdema. For Dr. Saundby tells us in his *Lectures on Bright's Disease* that Rehder experimented on five healthy persons for thirty-two days, and always found that they excreted by the kidneys 76·4 per cent. of the fluid imbibed, however great the quantity of that might be, that is, however much their blood might be diluted ; and in no case did œdema result, the lungs and skin no doubt sharing the excess of excretion with the kidneys. Further, Cohnheim and Lichtheim in numerous experiments on animals diluted the blood till its dried residue was only 11, 8, and even 4 per cent., instead of the normal 20 to 24, yet no anasarca resulted. Lastly, in a case shortly to be published, I injected 12 pints of water into



the rectum of a man suffering from general anasarca and epileptoid coma due to parenchymatous nephritis. The water was retained and the anasarca rapidly and almost completely disappeared. Here the injection of water and consequent dilution of the blood evidently aided the removal of the œdema instead of increasing it.

Anæmic blood has a smaller proportion of albumen than healthy blood, and this may have some effect in producing œdema. Immermann points out that the volume of the blood depends mainly upon the amount of albumen in the plasma, as albumen has the power to absorb water and incorporate it. Hence, the less albumen it contains, the less water will the blood take up from the tissues by osmosis, and therefore the water will tend to stagnate in the tissues as œdema.\* Becquerel and Rodier go so far as to state that dropsy must ensue whenever the proportion of albumen in the blood has fallen to from 8 to 6 per cent. I need not say that this is one of those precise chemical statements whose very exactness proclaims its untruthfulness. The body is no mere physical machine where we can trace the effect of a single cause uncomplicated by adjuvants or deterrents. We may grant that lack of albumen in the blood plays its share in the production of œdema, but it is only a share, for not seldom we find œdema absent when albumen is in very small quantity, while in other cases œdema is present though the amount of albumen is almost normal.

The condition of the corpuscles in anæmia can

\* See paper by Lazarus-Barlow, *Brit. Med. Jour.* March 1895.

scarcely have any direct action in the causation of œdema, as this varies so greatly in the different forms of anæmia, whereas the tendency to œdema is common to all, and but seldom varies directly with state of the corpuscles.

We may conclude, therefore, that the only morbid change in the blood which can directly cause œdema is its deficiency in albumen. Of the relative importance of this cause we know little, but I would remark that it is probably the drain of albumen from the blood which produces œdema in parenchymatous nephritis, and in some instances of severe diarrhœa.

A still more difficult question is the influence of the capillary endothelium on œdema. We can easily understand that the impoverished blood and the enfeebled circulation of anæmia may seriously deteriorate the condition of the endothelial cells, but morbid histology refuses to give us any proof that this does happen. Even in the case of inflammatory exudation I believe no change has been discovered by the microscope. I do not think any pathologist has described changes in the capillary endothelium as being the result of an anæmic state of the blood; but it may well be that an important deterioration of function may occur in so delicate a tissue without leaving any trace detectable by the microscope. Indeed, we know that changes in this endothelium will produce œdema, for we can observe this actually taking place in the processes of inflammation. But the changes brought about by anæmia must be very different to those arising from inflammation, for the character of

the two exudations differs widely. The œdema of anæmia contains far less proteid and, especially, many fewer white cells than does that of inflammation. [This difference between an inflammatory and a mere dropsical effusion sometimes proves of diagnostic value. For instance, the inflammatory fluid of peritonitis has a specific gravity of 1018 or more, while that of hepatic ascites is only 1010 and less; the ascitic fluid will not coagulate spontaneously, but the inflammatory one will. Hence if in a case of abdominal effusion we suspect chronic or subacute attacks of peritonitis are playing a part an analysis of the fluid will greatly help our diagnosis.] Inflammatory fluid can be exuded under considerable pressure, can even be forced into already tensely distended tissues; it seems to occur quite independently of tension; but the anasarca of anæmia is soft and pits easily. Moreover, anæmic œdema can be removed without any improvement in the blood, by merely changing the position of the part: position has comparatively little influence on inflammatory effusion. Now if the condition of the blood produced a morbid change in the capillary walls, and this morbid change induces œdema, why should this œdema vanish when no improvement has occurred in the blood, and therefore, presumably, no improvement taken place in the capillary wall. It seems evident that the capillary wall must play a smaller part, if any, in the production of anæmic œdema than in that of inflammation.\*

\* At the time of writing this I had not read Metschnikoff's Lectures. From these it would appear that in inflammation the

But we must remember that the capillary wall, even in health, greatly modifies the nature of the transudation which occurs through it. For instance, serum-globulin diffuses through a dead membrane much more slowly than does serum-albumen ; yet the proportion of these two substances in the fluid exuded into the lymph spaces outside the capillaries is the same as in the blood within these vessels. Another important fact showing that œdema is not only a filtration is this :—The constitution of all non-inflammatory exudations is almost the same, except in one particular, viz., the amount of proteid they contain : that is, as regards mere physical filtration (salts, etc.) there is no change ; but when it comes to osmosis, then the living element enters in. The proportion of proteid in hydrothorax is 2·8 per cent., in ascites 1·6 per cent., and in anasarca 0·35 per cent. only. In the two former, the osmosis has to take place through two membranes, viz., the capillary endothelium and that of the pleura or peritoneum, and this double osmosis greatly increases the proportion of proteid, and, what is stranger, the pleural endothelium is much more powerful in this respect than the peritoneal. Hence, we conclude that the endothelium modifies the character of the effusion, but does not itself produce it.

There remains the third cause—deficient cardiac action. That such does exist in anæmia is generally admitted now. The breathlessness on exertion seems to prove

capillary wall plays only a secondary part : that the leucocytes in great part come from the connective-tissue corpuscles : finally, that inflammation is purely a defensive process.

this, though Dr. Ingleby quotes so great an authority as Andral as endeavouring to show that this breathlessness arose because the blood in anæmia, being diminished in quantity, the lungs supplied this blood with more oxygen than was needed, the super-oxygenated blood producing disturbances which induced breathlessness ! A strange theory to us nowadays ! It is only in very severe cases that anæmic subjects become œdematous when lying in bed. Generally, at first at all events, the œdema occurs in the feet and legs, and is worst at night ; on rising in the morning it is often gone, and is always much less. What has produced this improvement ? The quality of the blood has undergone no change, nor have the tissues external to the capillaries altered. We cannot, therefore, understand why the nutrition of the capillary endothelium should have improved. But there has been a great change in the mechanical conditions of the circulation ; before, the blood had to be pumped up some 4 ft. from the feet to the heart ; now, in the supine position, it has merely to be driven along a level way. This is the great, in fact the only difference that has arisen. It is true that the improved circulation may benefit the nutrition of the capillary endothelium by supplying it with a larger quantity of blood. But it will be an increase in quantity, not quality, and variations in mere quantity do not seem to have much power in inducing or removing œdema. No doubt the comparative stagnation of blood which has accompanied and preceded the œdema has had an injurious effect upon the endothelium as well as



upon the other tissues situated in the œdematous region, and this injurious effect may have aided the œdema. But on the other hand, this morbid change in the endothelium may be one which tends to *prevent* exudation, and so hinder the production of œdema, rather than cause it.

The whole question of anæmic œdema may be summed up thus:—In the mild and ordinary form, which is usually confined to the legs, cardiac debility,\* which under the influence of gravity allows stagnation of the venous blood, is the chief if not sole cause; in the severer forms, which arise while the patient is lying supine, where exudation occurs in various parts of the body, including the large serous cavities, the plasma's deficiency of albumen is no doubt an additional cause, and the deterioration of the capillary wall may also to some extent assist, though this last is mostly a matter of conjecture.

## LECTURE II. THE HEART IN DEBILITY

To-day I shall consider the changes which take place in the heart. These have to be viewed in two aspects: (1) the deviations from normal which are

\* Cardiac debility is an insufficient phrase. Feeble circulation in its widest sense would be more appropriate. For want of tone in the deep muscles, and especially in the small fibres and elastic elements of the skin and subcutaneous tissues, must also have great influence in emptying the lymphatic spaces. Anæmic œdema is not so much the result of excessive exudation into the tissues, but is rather due to the atonic inability of these to pass on to the lymphatics and veins a normal amount of exudation.

to be observed clinically ; and (2) the actual morbid changes which bring about these clinically-observed deviations. Even regarding mere clinical observation there is much difference of opinion ; and when we come to discuss the conditions which produce these, we at once enter the vague confines of the unknown.

The alterations of the heart are marked and characteristic ; they involve the position and nature of the apex beat, the area of dulness, the occurrence of systolic and diastolic murmurs, the accentuation of the pulmonary and aortic second sounds, and great instability of cardiac action.

Before discussing these, I will give my reasons for using the **superficial** rather than the **deep** cardiac dulness notwithstanding that it is this latter which is thought far more highly of by most authorities.

They are these : (1) To percuss out the deep dulness accurately, a firm and what I may term a severe stroke is necessary ; such a stroke as is extremely painful in all neuralgic conditions of the heart—a stroke which may give a most injurious shock in dangerous maladies like pneumonia, pericarditis, or pleural effusion ; and yet it is in these states that an accurate knowledge of the size of the heart is most valuable.

(2) It is more difficult for the senses of touch and hearing to appreciate slight differences in resistance and quality of tone when the pressure and the quantity of sound are great than when they are small. If your finger is pressed upon by a pound weight, it may be hard to

detect the addition of an ounce ; whereas if the original weight had been an ounce only, the addition of another would at once have been manifest. Similarly, if a blind man is endeavouring to detect the outline of a solid object, he passes his fingers over it with great delicacy. So it is with sound : the greater the quantity the harder it is to appreciate small differences of pitch and *timbre*. Hence the severer the percussion note, the harder will it be to appreciate the value of that portion of the resulting resistance and sound which is due to a slight abnormality in the sounding board.

(3) In cases where solid lung, new growth, or pleural fluid adjoins the heart, any attempt to obtain an accurate map of the deep dulness is impossible. In such it is sometimes extremely difficult, if not impossible, to distinguish the *superficial* dulness of the heart from that of its neighbouring dulness, the notes and resistances of the two are so nearly alike ; and if one of these similarly sounding bodies be placed below the other, and we have to distinguish its qualities by percussing it indirectly *through* the other, it is evident that this difficulty is very greatly increased.

(4) The arch of the aorta reaches up to the first rib : the true deep dulness must therefore also extend to this. How, then, from estimation of the deep dulness shall we learn if the heart enlarges upwards ; or if we could learn it, how might we express it ?

(5) The deep dulness of the liver reaches up to the fourth space ; when percussing out the deep cardiac dulness on the right in the fourth space we shall

therefore be confused by this liver note. And the right dulness in this fourth space is perhaps the most valuable of all to obtain ; it is our only physical landmark of enlargement on the right, while on the left the accurate estimation of the apex beat prevents us from going very far wrong.

(6) Percussing out the deep dulness requires considerably more exposure of the chest than the superficial. Any exposure which is not absolutely necessary is always to be avoided in women, and in private practice when your patient is dressed it will often prove a very serious obstacle to the accurate estimation of the deep dulness.

(7) The deep dulness is said to be the more accurate in emphysema. I fail to perceive the truth of this, The fringes of the lung are the parts most liable to this degeneration. Hence, on percussing from pure lung to where the deep outline of the heart commences we are percussing from a portion of lung with normal resonance to a part which has the hyper-resonance of emphysema. It is easy to see how the commencing deep dulness of the heart *plus* the hyper-resonant fringe of emphysema may give forth a note very nearly approaching that of normal lung. It is true the note is not of quite the same quality ; but neither is it the note of the ordinary deep cardiac dulness, and any but a very sensitive and experienced ear will probably be confused by it and draw an uncertain conclusion. In fact, we have to make an inference from our past experience, from the symptoms, and from all the physical signs together,

and form a judgment thereon. And this is the very thing we have to do regarding the superficial dulness. We have to act in the same way with deep-chested and large-lunged individuals. Here the whole thorax is more resonant than usual. There is no dulness so absolute as the superficial cardiac dulness in the normal individual, nor is the partial dulness over the deep outline of the heart so distinct as it is wont to be. We have to be content with a less dull note in each case, but it is easier to determine what this note shall be in the superficial than in the deep dulness as our method of investigation is more delicate.

(8) It is said the superficial dulness, when accurately obtained, gives you no definite knowledge of the exact outline of the heart. In one sense this is true enough ; but the exact outline of the heart is not what we wish to know. We want to find out whether the heart is enlarged, the degree of this enlargement, and whether this be chiefly upwards, downwards, to the right, or to the left. These facts, after we have once fixed the position of the apex beat, the superficial dulness will determine for us quite as accurately as the deep. Indeed, after having in numerous instances percussed out as carefully as I could both dulnesses, I have never found that the superficial dulness has ever been anything but a faithful guide both as to the amount and nature of the enlargement which the heart has undergone, and I have not found cause to make any correction from my subsequent determination of the deep dulness.

(9) Finally, we are all imperfect mortals. Often we



grow weary and often we are pressed for time ; there is no doubt that the superficial is by far the easier and also the swifter dulness to determine. No one will gainsay that it is better to define accurately the superficial than determine the deep in a hurried and confused manner. Moreover, I am afraid that the best of us would sometimes omit percussing the heart altogether if we trusted to deep dulness only—partly from the pain the examination causes, partly from the exposure it necessitates, and partly from the difficulty of its precise determination. And this, “sometimes” would occur most frequently in those very diseases—for example, pneumonia and pericarditis—where it is of the utmost importance daily to obtain correct information of the variations in the size of the heart.

Believing as I do that, from a therapeutic point of view, cardiac percussion is of more value than cardiac auscultation, and that it should be made use of at least as frequently, these several objections to obtaining the deep dulness seem to me to be of great and serious moment, and, moreover, to be thoroughly real and practical ones. Do not mistake me. I am not for one moment wishful to depreciate the value of the deep dulness as an adjuvant after you have determined the superficial ; but in ordinary practice we usually only have the opportunity of fixing one of these. When this is so, then I believe on the whole the determination of the superficial will prove the more valuable.

To return to the consideration of the changes in the heart :

(1) **The Apex Beat** is displaced upwards and to the left, usually being in the fourth left space nearly as far out as the nipple line, the displacement to the left being thus very little, if at all, in excess of what is required by the upward movement, if we consider the apex as a point travelling along the arc of a circle whose centre is the aortic valve. It is rarely displaced downwards, and I can only find one record of its being in the sixth space, though it is not seldom in the fifth, in which case it may extend, even as much as an inch, beyond the left nipple line. Its impulse is diffused, feeble, and sudden, unless during mental excitement, when force may replace feebleness. This feebleness may be so great as to render localisation impossible without the stethoscope. But there is another very evident impulse, chiefly seen in the second and third spaces, but often in the fourth and first. It spreads from above down and from right to left, with rapid vermicular movement. In each space it starts close against the sternum, but in the third and fourth it is frequently seen to the right of this bone ; in rhythm it is one continuous movement, occupying the whole period of the auricular and ventricular contractions.

(2) **The Area of Superficial or Absolute Dulness** is not increased to the left any more than is necessitated by the position of the apex. Its increase to the right is very small, rarely reaching beyond the mid-sternum, but its increase upwards is remarkable, often reaching to the second space. It is this *upward* enlargement to which I particularly wish to draw your attention, as it

gives us our greatest help in diagnosing functional from organic mitral disease, in this latter the enlargement of dulness being more to the right than upwards.

(3) **There are three Systolic Murmurs**, which I will term (anticipating my view of their causation) pulmonary, tricuspid, and mitral. The focus of the **Pulmonary** murmur is the junction of the third left cartilage with the sternum ; it is best conducted along a line leading thence up and out to the left shoulder, and it is not well heard to the right of the sternum. The **Tricuspid** murmur is heard best over the third and fourth left spaces, but also well to the right of the sternum, and, often with marked distinctness, upwards to the second right space, and even some way along the vessels of the right side of the neck. You thus perceive that I do not think it necessary to consider a murmur heard in the last situation to be aortic in origin. I have always been able to explain its existence as arising from malfunction of the pulmonary artery or tricuspid valve. As regards the latter, we must remember that the right auricle in anæmia not seldom occupies the second right space, and one can see no good reason why the audible vibrations of tricuspid regurgitation should not be conducted as far as the internal jugulars. Then, as to the pulmonary artery, it gives large branches to both apices of the lung ; hence the audible vibrations of its murmur may be heard over these ; also we must not forget that the pulmonary artery and aorta are closely united, each being tensely distended with the same fluid, though certainly at different pressures ; still, one can

scarcely imagine that vibrations arising in the one would not be transmitted to the fluid in the other, and thus a pulmonary murmur would be heard along the aorta and its branches, and so simulate an aortic systolic murmur as well. Similarly the murmur of aortic regurgitation is often best heard in the third left space, that is, at a point where its vibrations must reach the ear through the superimposed pulmonary artery. Both the tricuspid and pulmonary murmurs are increased on lying down, but the pulmonary much more than the tricuspid, and moreover the increased area over which the tricuspid is heard is usually in a downward direction on both sides of the sternum, but that of the pulmonary is outwards and upwards to the left shoulder. Both murmurs may be only audible in the supine position, though plainly so then. With tricuspid murmur there is usually distinct systolic pulsation in both jugular veins on the right side of the neck, though I do not think this necessarily exists, and when it does it has to be carefully distinguished from a venous pulsation which I shall speak of as frequently occurring apart from tricuspid regurgitation. The **Mitral** murmur is best heard at the apex ; it is soft and not well conducted, and seldom entirely replaces the first sound. Should it, however, be loud and harsh, it is quite as well heard in the axilla and at the angle of the scapula as the murmurs due to organic mitral disease. It is the most fugitive of the three, sometimes occurring with every third or fourth beat only, and seldom persisting for many days together, but coming and going with unaccountable vagrancy.

(4) Far rarer than these systolic murmurs are those which are **Diastolic** in rhythm : yet they occur. The commoner is one occupying the mitral area : it is soft and post- or mid-diastolic in time, never, so far as I have noted, being presystolic either in time or character. The other occupies the tricuspid area and is identical in time with the mitral.

5. **The Accentuation of the Second Sound of the Heart at the Left Base**—that is, over the pulmonary valves—is nearly universal in all cases of anæmic debility which are up and about ; sometimes it is absent in those confined to bed, but it usually becomes evident on their getting up. The accentuation of the aortic second sound is less marked, frequent and persistent.

6. **The Cardiac Action is in a Condition of very great Instability** ; the least disturbance, mental or physical, is enough to very seriously change its rhythm ; palpitation in more or less degree is very common. But though the sudden initiation of exertion may thus upset the heart, yet on the moderate continuance of such exertion the rhythm may improve, showing that the original disturbance was not entirely due to absolute muscular feebleness.

Illustrations of the truth of these remarks might be multiplied indefinitely, but I will content myself with giving a few instances occurring in various varieties of anæmia.

**Chlorosis.**—Jemima W., 19, single, sewing machinist. Her heart's apex beat in the fifth space, one inch to the left of the left nipple line. The dulness was absolute up to the third



cartilage and almost so right up to the clavicle. Absolute dulness did not extend beyond the left edge of the sternum on the right. There was a systolic murmur over the pulmonary area, and another became evident at the apex, this latter being well conducted to the axilla. The pulmonary second sound was much accentuated.

**Chlorosis with much Soft Œdema of Legs.—**

Sarah S., 18, single, servant. Her heart's apex beat in the fifth space and nipple line. Absolute dulness reached up to the second cartilage and on the right as far as the mid-sternal line. The pulmonary second sound was strongly accented. The external jugular vein filled from below and pulsated distinctly. There was no murmur anywhere in the erect posture, but on lying down a short systolic one was heard over a narrow oval area whose long axis reached from the fourth left cartilage up and to the left into the outer part of the first left space. The pulse was 74, of fair size, rather hard and sudden.

**Simple Anæmia.**—Ada B., residing at home, single, 20. The heart's apex beat in the fifth space and the nipple line. Absolute dulness reached to the third cartilage and the middle of the sternum, but there was partial dulness up to the clavicle. At the apex was a systolic murmur conducted into the axilla. A systolic murmur was also heard over the pulmonary area and was conducted up towards the left shoulder and the inner part of the right first space. A murmur and a thrill—both systolic—existed over both jugulars on both sides of the neck. There was a loud venous hum in the intra-clavicular fossa. On lying down the venous hum became less marked, the apical murmur remained unchanged, the basal murmurs were much increased in loudness, and a systolic murmur was well heard over the lower part of the sternum and to the right of this bone.

**Anæmia with early Phthisis at the Right Apex.**

—Carrie M., teacher, single, 22. The apex beat was in the normal situation. There was a systolic murmur in the third left space, on lying down this extended down and across the

sternum. The pulmonary second sound was much accented. A *bruit de diable* was heard (on pressure only) over the internal jugular veins only.

**Anæmia with frequent Faintness.**—Eliza B., 17, single, French polisher. Apex beat in nipple line and fourth space. Absolute dulness up to third cartilage and to mid-sternum; partial dulness reached to clavicle. At the apex was a soft systolic murmur conducted slightly towards the axilla. No murmur at the bases, but a systolic one was heard in the vessels of the neck. The heart's action was regular and the impulse forcible. The pulse small, fairly sustained, and of moderate tension.

**Debility.**—Emily D., 24, single, clerk. Apex beat in the fourth space and nipple line with a systolic thrill. Absolute dulness to the third cartilage and left edge of the sternum. A pulmonary systolic murmur existed in the second left space near the sternum and reached up to the outer part of the first space. The area of this murmur was increased on lying down. There were venous murmurs in the neck.

**Debility with Bronchial Catarrh.**—Mr. B., 19, an artist. His apex beat was in the fifth space and nipple line. Absolute dulness reached to the second cartilage and the right edge of the sternum. The action was excited and the impulse widely diffused. Pulmonary second sound slightly accented. On lying down a systolic murmur arose which was best heard in both second spaces and radiated from these (? pulmonary or tricuspid or both). Pulse was quick, but fairly strong.

**Debility with irritable Cough and Fibrosis at the Right Apex** (? from old pneumonia).—Mr. J., medical student, 20. Heart's apex beat at the lower edge of the fifth space in the nipple line. Its impulse was forcible and diffused with a slight thrill. The second pulmonary sound was much accented and a systolic murmur appeared in the pulmonary area on his lying down.

**Debility.**—Mr. H., 27, single. Apex beat in normal situation. First sound slapping. Absolute dulness to third cartilage and mid-sternum. The second pulmonary sound was accented. A systolic murmur existed in the second left space extending up and out towards the shoulder, and was also feebly heard in the third space. Occasionally another systolic murmur was heard at the apex and conducted slightly towards the axilla.

The superficial dulness of a heart constructed by tabulating the points above alluded to and taking the average value of each is as follows: apex beat at the lower border of the fifth cartilage and in the nipple line, the absolute vertical dulness reaches up into the lower part of the second left space, but on the right the absolute dulness does not quite extend to the mid-sternal line.\* A pulmonary murmur was heard in eight out of nine cases; the pulmonary second sound was noted as accented in six, no note being made of it in the other three; there was an apical systolic murmur in four, and a tricuspid in three only, but the jugulars filled from below when distally compressed in one case where no tricuspid murmur is recorded.

I have given you, I think, a truthful representation of the heart as it appears to us when we examine the chest in the subjects of anæmic debility. There now remains for me the far harder task of laying before you the causes of these abnormalities; they should be such that one in no way fights against another, but

\* In the accompanying diagram the continuous outline represents the area of superficial dulness above mentioned, whilst the small dotted circumference indicates that of a normal heart and the larger

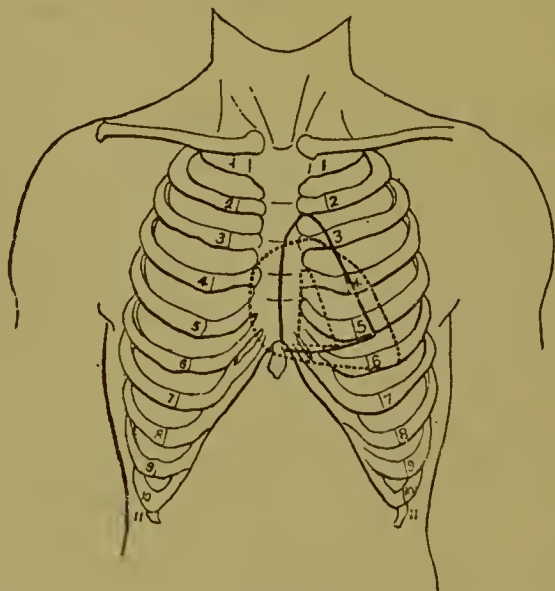
rather should they be mutually interdependent, with logical coherence.

There are two main conditions which underlie the whole question ; these are, first, the left ventricle

dotted area represents the outline of a fairly typical case of organic mitral disease.

The reason why the **right auricle** enlarges in organic mitral disease and not in anæmia is, I think, this : in anæmia the strain on

FIG. 5.



the right side is not constant, but only during exertion, and there are long spells of recuperative rest. During the exertion the buffer-action of the right ventricle is able to save the right auricle and great veins from any serious strain ; during the ensuing rest and, moreover, with the too-small quantity of venous blood flowing through them these organs are able to regain their tone. But that they *do* feel the strain of exertion is evident to any one who has noted the neck-veins of a chlorotic after a hurried run upstairs. We must remember too that the strength of the left heart is more than normal in organic mitral disease and less than this in anæmia. So that the difference in tension of the regurgitant blood must be very great.

undergoes little, if any, enlargement ; and, secondly, the right ventricle always enlarges, and this, not as a secondary result, but as the chief and primary cardiac share in the anæmic changes. I am aware that this is as yet somewhat heterodox doctrine, but the more experience I have of these cases the more convinced am I of its truth. I believe the initial strain falls upon the right ventricle, the work of which is considerably increased ; as a result of this it partly dilates and partly hypertrophies, and performs more work, though not sufficient to keep the pulmonary circulation up to its normal state and to overcome the failure which usually takes place in the left ventricle. I will now consider what *a priori* reasons there are for taking this view of the matter, and afterwards I will try to show that, granted this view to be correct, it will fully explain the signs we have been discussing.

Anæmia is no local disease ; the body fails as a whole, and the heart muscle no more than other muscle, nor the heart nerves more than the nervous system generally. The whole constitutional state is below par, and every action, mental and physical, the beating of the heart included, is performed feebly ; but there is no evidence to show that the feeble left ventricle is unable to supply the diminished wants of the feebly acting body. Secondly, the left ventricle is, as the small pulse of anæmia incontrovertibly tells us, unable to send on a normal amount of blood. This is probably merely an incident in a general feebleness of the circulation, but if it be owing to its inability to propel the



amount which the right ventricle is anxious to send it, yet this does not necessitate its enlargement; it may simply adjust matters by but partially emptying itself, and then refusing to admit the full quantity of blood from the lungs, for the wants of the anæmic body are not imperious, and it will only too easily content itself with a smaller amount, and so lead to further anæmia. It is continuous toil which affects the left ventricle—witness the irritable heart of the soldier after forced marches, and that of the blacksmith; now continuous toil, mental or physical, is an impossibility to the anæmic patient.\*

But anæmic people who are up and about and trying to do their work in the world, have a certain standard of speed and persistence set them by the healthy people they see around them. This standard they try to attain; they therefore start off with the vigour of a healthy person, but their feeble muscle or nerve cells soon pull them up and they have to rest, starting off again in a few minutes with more than normal vigour,

\* It is true that when there is high tension in anæmia the left ventricle has extra work to do; but most of these high-tension anæmias are not severe ones, while in nearly all the debilities of middle life the condition is one of low tension. Moreover, if the tension is high and the ventricle too feeble to fight against it, there is no need for it to enlarge. It may content itself with a still more imperfect systole, and so still further handicap the right ventricle, both by raising the tension in the pulmonary circuit and also by more feebly supplying the body (including the right ventricle) with arterial blood. Further, the cause which produces high tension in the systemic circulation will probably induce it in the pulmonary circulation also, so that the right ventricle will suffer equally with the left in this particular.

to make up for lost time, but only the sooner to be re-arrested by helpless debility. Their work is therefore done in jerks, the toil during the jerk being far beyond their strength. They might perhaps do just as much in the aggregate without injury to themselves if they worked from beginning to end at a steady, slow rate commensurate with their strength ; but the forces of imitation and emulation are too strong for them, and they persist in exhibitions of normal energy with subnormal bodies. But even had they perfect control of themselves, how can they avoid sudden efforts of high pressure ? The anæmic school-girl standing up in class has to concentrate her brain power to answer with costly speed the question rapidly passed down from one to another, or has to work sums for marks against time. The housemaid is bound to run upstairs quickly to answer her mistress's bell, to carry trays full of food, and scuttles full of coal. This quickness of answer, these trays and scuttles have been formed for healthy persons ; to them they would act but as a sturdy developmental stimulus, but to the anæmic they become a breathless and exhausting labour. If anæmics held sway over toil there would be no quickness of performance, no strenuous effort allowed. Luckily for the world's progress, but unluckily for them, they have to play a very subordinate part on life's stage, and to be content with things as they find them.

It is thus to the dangers arising from sudden transitory toil that the anæmic person is peculiarly liable. The first effect of exertion is an increase in the

respirations, and very little exertion is sufficient to do this. This increase in the respirations can only mean that exertion at once makes a demand for more oxygen. Extra oxygen can be supplied in two ways ; by increasing the amount of it taken up by each red corpuscle, or by increasing the flow of blood through the lungs. In anæmia the oxygen capacity of the red corpuscles is peculiarly small and the number of the corpuscles themselves is diminished ; hence the anæmic endeavours to meet the demand for more oxygen by increasing the rate of flow through his lungs ; to do this he calls upon the right ventricle to work with more than its normal force. Thus the initial strain is thrown upon it. This causes it to enlarge, and its enlargement, as we have seen, is unaccompanied by corresponding enlargement of the left ventricle.\*

If this position be granted me, it is easy to explain :—

(1) *The Apex Beat*.—I have said that it is so displaced that the line† from the aortic valves to its new position is of much the same length as the line from the same valves to the normal apex beat—that is, the length of the left ventricle is unchanged. Its feebleness is partly due to feeble action from insufficient emptying, as shown by the small pulse, and partly to its not coming so close against the chest wall owing to the enlargement of the

\* We know little of the vasomotor control of the pulmonary system, but it may well be that some special constriction occurs to hinder the ingress of corpuscles so peculiarly unfitted to be absorbers of oxygen as the feeble, hæmoglobin-lacking ones of anæmia.

† Even when the length of this line is increased, it does not

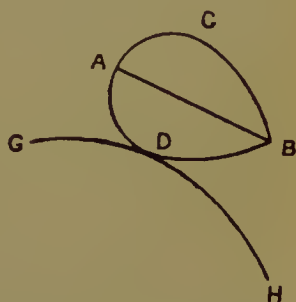
right ventricle. The right ventricle may indeed entirely occupy the apex, so that the impulse of the left is not perceived at all. (It is for this reason that the pulse is a much surer guide to the state of the left ventricle in these cases than the apex beat.) The other vermicular impulse spoken of is over the body of the right heart, and shows the movements during systole of this ; it is more evident often than the apex beat, because, as we shall see directly, the right ventricle dilates chiefly at its base and thus tends to prevent the apical impulse reaching the chest wall ; it is vermicular because the right ventricle is straining its utmost, just as the movements of a man who is trying to vault over a bar placed high above him, at the utmost limit of his vaulting power, are of the same vermicular character.\*

necessarily follow that the left ventricle is enlarged : the increase may be due to the fact that the apex beat is formed by the *right* ventricle. (*Vide Case 5, infra.*)

Let A=site of aortic valves ; the fixed point or fulcrum, B=apex beat ; ACB=left ventricle, and ADB=right ventricle ; GH=diaphragm. Then if we consider GH a fixed plane and AB a fixed length, it is evident that the arc ADB can only lengthen by forcing B up and out.

\* That part of this impulse is auricular-systolic in time—*i.e.*, occurs before the apex beat—I have no doubt. But it is not caused by either auricle, but by the dilated conus arteriosus, for (a) it is confined entirely to the left side of the sternum (hence not due to the right auricle), and (b) the left auricle in these cases is as completely behind the pulmonary artery as in the normal heart.

FIG. 6.



(2) *The Dulness*.—This is due to a dilatation of the right ventricle and pulmonary artery, unaccompanied by a corresponding increase in size of the left ventricle or right auricle. Hence the right ventricle's dilatation takes place chiefly in the upward direction, as it is here freest from the restraining influence of the stout left ventricular walls. The two ventricles may be regarded as a cone; if one half of this be rigid (left ventricle) and the other half uniformly distensible, it is evident that with a uniformly distending force the chief dilatation of the distensible side will occur about the free semicircular border of its base, and that portion of this border which is connected with the pulmonary artery dilates most, as it is farthest from the restraining influence of the inferior vena cava.\* But the excess of dulness is partly caused by dilatation of the pulmonary artery itself; we must remember how easily this is enlarged by an increase in tension; for instance, whereas the capacity of the rabbit's aorta is only quadrupled by an increase of pressure from zero to 200 mm. of mercury, the same result is attained in the pulmonary artery by merely raising the pressure from zero to 12 mm. The accentuation of the pulmonary second sound in anæmia shows us that the tension in this artery is increased; and as the usual tension in it is not great—some 70 mm. Hg.—this increased tension will assuredly be appreciated by the artery and

\* It will be remembered that this vein opens into the auricle close to the lowermost point of the auriculo-ventricular septum and immediately after its passage through the firm, tendinous diaphragm, which is practically immovable at this point.



show itself in inducing its dilatation. But that this peculiar form of dilatation really does happen we have the best of all proofs in the condition found post mortem in those who have died from debilitating disease or from a disease such as pneumonia, which throws special strain on the right ventricle. On this point I have made the following observations in the post-mortem room of the Queen's Hospital :

CASE 1.—Death from tuberculous peritonitis. The heart's apex lay beneath the fourth space in the left nipple line. The pulmonary valves were beneath the lower border of the first left space, the whole of them being to the left of the sternum. The right appendix extended for one-third of an inch into the first left space. The left ventricle was firmly contracted; the right flaccid and filled with fluid clot.

CASE 2.—Death from hepatic carcinoma. The heart's apex lay beneath the sixth rib, midway between the nipple and anterior axillary lines. The pulmonary valves lay beneath the middle of the second left space.

CASE 3.—Death from croup and pneumonia. The heart's apex lay beneath the upper edge of the sixth rib in the left nipple line. The pulmonary valves were beneath the second left cartilage.

CASE 4.—Death from carcinoma. The heart's apex lay beneath the fifth space and in the nipple line. The heart reached to the right edge of the sternum. The pulmonary valves lay beneath the upper edge of the second cartilage.

CASE 5.—Death from tuberculous phthisis. I did not see this heart *in situ*, but after removal I found the heart's apex was formed by the right ventricle, which reached one-eighth of an inch below the left. The left was fairly contracted, but the right dilated. The posterior wall of the pulmonary artery (as shown by the relative heights of the upper attachments of the

adjacent valves) was a good third of an inch above the aorta ; while the upper attachment of the anterior pulmonary valve was a good half-inch above the upper attachment of the posterior one—that is, five-sixths of an inch above the aorta. This case shows that the anterior wall of the pulmonary artery is pushed up more than the posterior. This condition was found in the empty dead heart ; when it was full and living the difference in the position of the valves would have been much more marked.

I would also refer you for proof of this high range of dulness to some post-mortem reports on anæmia in Dr. W. Russell's most valuable book, entitled "Investigations into some Morbid Cardiac Conditions," which he published in 1885.

This pushing up of the pulmonary valves shortens the distance between the extremities of the pulmonary artery, and so renders its dilatation easier.

(3) *The Murmurs*.—The pulmonary murmur is due to a complicated change in the shape and position of the pulmonary artery, which constricts it at a point, and thus gives rise to a fluid vein and its sonorous eddies. This change consists in :—(a) As we have seen, the pulmonary valves are carried upwards till they are vertically over the bifurcation, which is practically a fixed point. This necessarily brings the valves nearer the bifurcation—that is, the two ends of the artery approximate each other, and therefore the curve of its circumference is a sharper one. This carrying up of the vessel presses its superior wall against the hollow of the aortic arch. Now the tension in the aorta is more

than double that in the pulmonary artery, and its wall is correspondingly stouter ; hence it may be looked on as a rigid body. The pressure of the pulmonary artery against it will therefore flatten the superior wall of the former.

(*b*) Owing to the peculiar one-sided dilatation of the ventricular heart, the plane of the right auriculo-ventricular septum ceases to be in the same plane as the left, but becomes tilted from left to right upwards and backwards. This will prevent the axis of ventricle and artery being in the same straight line, and hence the blood during systole will impinge upon the side of the artery, and hence will arise eddies. This condition is still further increased by the union of the pulmonary artery to the aorta, which will not allow the posterior wall of the former to rise to anything like the same extent as the anterior one (*vide* Case 5, *supra*). (*c*) It follows from this that the anterior semicircle of the arterial coat is also more pushed up than the posterior, and must therefore become more lax ; hence an additional reason for its compression against the aortic arch. (*d*) The movement of the apex to the left and somewhat upwards still further alters the angle at which the ventricle lies to the pulmonary artery. This movement of itself, when of considerable degree, is sufficient to produce a murmur, as we know from the temporary murmurs arising in pleuritic effusion and which vanish at once on the withdrawal of the fluid. In the horizontal position the murmur is increased because the action of gravity ceases, the right ventricle therefore forces the blood into the artery with more force. This

of itself would increase the murmur, but there is also no longer the weight of the right ventricle to stretch the artery lengthwise; hence this tends to dilate still more and to rise up still further against the aortic arch.

The tricuspid murmur has for its causes :—(a) The increase in the circumference of the valve ring, which is one result of the enlargement of the right ventricle; this is more especially the case as the valve ring is situated at a portion of the right ventricle which we have seen is liable to disproportionate stretching. (b) Roy and Adami have shown that the muscoli papillares act independently of the heart walls; further, that with high tension the contraction of the wall is delayed, but the papillary contraction is as sharp as ever. In anæmia the tension in the right ventricle (as shown by the accentuation of the pulmonary second sound) is increased, the ventricle is also dilated; there is, then, a double reason why the contraction of the wall should lag behind that of the muscoli papillares. The too-soon contraction of these latter may at first pull the valve flaps too far down into the ventricle and so allow of regurgitation, especially as the bases of the papillary muscles are farther than normal from the valve plane, owing to the enlargement of the ventricle. (c) If (a) and (b) allow of any outflow, then the change in the angle of the valve plane will tend to produce a murmur as in the case of the pulmonary valve. (d) Hesse and Ludwig have shown that the circumference of the base of the heart during diastole is twice that of its circumference during systole. This change is due to

muscular contraction. In debility it seems more than probable that this contraction is less marked—that is, the auriculo-ventricular openings are less diminished in area than is the case in systole of the normal heart. Here then would appear to be a further cause of regurgitation. As regards the effect of the horizontal position, this is due to gravity, as in the case of the pulmonary murmur, but as the pressure in the right auricle is much less than in the pulmonary artery this force will have a greater proportional effect.

The mitral murmur can but seldom be due to dilatation of the left ventricle, as I have shown that this seldom occurs to any considerable extent. Its most frequent causes are : (*a*) lack of due contraction of the valve ring (*vide* (*d*) under tricuspid) ; (*b*) want of due co-ordination between the muscoli papillares and the ventricle wall. This latter has been shown to occur by Roy and Adami with large doses of strophanthus and they suggest that in certain diseased conditions it may also arise. This inco-ordination may act in two ways ; it may cause regurgitation at the commencement of the systole, by shortening the muscoli before the ventricle has shortened, or by the muscoli finishing their contraction too soon and lengthening when the ventricles are shortest, and so inducing regurgitation by allowing the valves to penetrate too far into the auricle. Probably this latter inco-ordination is the more common, for very often a short systolic murmur is heard following immediately the first sound and before the second.\*

\* The causes of the diastolic murmurs I do not as yet compre-



That these murmurs are not due to changes in the blood is sufficiently shown : (1) If the blood is largely diluted with water, murmurs arise, but these then occur at numerous points of the vascular system, and show no special fondness for the localities in which they arise in anæmia. (2) Murmurs appear in cases of debility where very small or no change is apparent in the blood, and yet they may be absent even in severe cases of chlorosis and pernicious anæmia. (3) The intractability of the disease seems in no way to depend upon their presence or absence. (4) Persons convalescing from acute disease—for example, typhoid—and suffering from anæmia, may have no murmur so long as they are confined to bed, but yet on being allowed to get up and undergo physical exertion, murmurs will arise.

4. *The Accentuation of the Pulmonary Second Sound.*—This must be due to high tension in the pulmonary circulation. This high tension is due to the feebleness of the left ventricle, which causes it only very partially to empty itself during systole : hence, there is not room for so much blood to enter from the auricle during diastole as normally ; thus the tension in the pulmonary circuit is raised. Fresh equilibrium might be restored by the right ventricle similarly refusing to empty itself, were it not that in anæmia there is, even with a normal circulation, a poor supply of oxygen to the tissues on account of the poverty of the red corpuscles, hence, any diminution of this at once produces “air hunger.” This

hend. Dr. Stacey Wilson (*Lancet*, September 15, 1894) has made a most ingenious suggestion, which, however, I am unable to accept.

produces a reflex increase of respiration. With every expiration blood is forced from the lung towards the pulmonary veins and the pulmonary artery ; on the left side it finds its way into the left heart and the systemic circulation, but on the right the pulmonary valves prevent such outlet, hence any increase in rate or depth of the respirations raises the blood pressure in the pulmonary artery. So that the benefit derived from the increase of respiration be not lost, the right ventricle must rise equal to the occasion and strengthen its contraction so as to deal with the increased pressure in the pulmonary artery. Indeed, just as an increase of vasomotor contraction in chronic Bright's disease increases the contractile force of the left ventricle, so does an increase in the blood pressure in the pulmonary artery increase the contractile force of the right. It is the instinct of the right ventricle to keep the oxygenating power of the blood at its normal level ; to do this it will strain every effort, as I have before explained ; and any of those temporary exertions which we all so frequently yet unconsciously undertake will only increase the necessity for renewed exertion on the part of the right ventricle and, therefore, the tension in the pulmonary artery.\*

(5) *The Instability—Irritability, if you will—of the Cardiac Action* is due in a great measure to feebleness of the cardiac muscle ; in some cases fatty degeneration is found. The normal work of the body becomes thus a

\* Consider also the possible increased vasomotor tonus in the pulmonary system : *vide* pp. 342 and 344.

toil only just able to be performed by the heart ; any slight increase may tax its strength to the utmost. Compare the feeble heart for a moment with a donkey who is dragging a load almost beyond its strength ; if the cart wheel happen to go over any small unevenness, such as a loose stone, the poor animal is almost upset with the sideward jerk of the cart shaft. Again, if there be even a slight gradient, it will have to strain its utmost ; but if it had been harnessed to its own light coster-cart, this slight gradient and loose stone would have been merrily, almost unconsciously, dealt with. The origin of breathlessness and palpitation in anæmia arises from similar causes ; the heart is harnessed to a body capable of actions beyond its strength. Yet this is not all ; for we sometimes see anæmics become breathless with some sudden slight exertion or emotion, who when the first surprise is over settle down to their new condition and work well and steadily to the end. Here the feebleness could not have been in the muscle ; but rather in the manner in which the fresh command for exertion was delivered to the muscle ; the nervous organisation must be at fault ; perhaps with the hyper-sensitiveness of neurasthenia, which prevented the message being delivered with due toning down and modification.

During adolescence there is another important cause of heart failure apart from any anæmia. I refer to the rapid increase in size of this organ at that period. In the *British Medical Journal* for Nov. 27, 1886, Dr. Pitt has referred to the investigations of Prof. Beneke, which

show that between seven and fourteen years the annual growth in the heart is 8 per cent. of its size, but that during the changes which accompany puberty it increases 80 to 100 per cent. Now in healthy, well-fed and cared-for children the changes of puberty may occur in one year ; the amount of cardiac change required in that year will thus be enormous ; the heart has to double its size. It is easy to understand how with such immense developmental changes occurring in it, it may prove functionally unable to perform the physical exertions which the active life of a young boy or girl demand ; hence cardiac dyspnœa arises in apparently quite healthy and robust young people. In the same paper, Dr. Pitt has shown that the changes in the heart are, in the main, identical with those which I have described.

I feel that in the foregoing description of the anæmic heart I have chiefly borne in mind the heart of adolescence and young adult life. It is true that as life gets older the left ventricle tends more and more to become involved ; indeed, at and after fifty one very rarely comes across a case of uncomplicated weakness of the right ventricle. This is because in the ordinary stress of existence the left ventricle bears the lion's share and oftenest shows signs of this in degeneration of its tissues. Hence, when the peculiar strain of anæmic debility arises it is less able to withstand it than the right ventricle. But this in no way invalidates my position that in this condition the main strain falls on the right and that, if the whole heart be previously equally healthy, it is this ventricle which will chiefly, if not entirely, suffer.



## LECTURE III. VENOUS CHANGES—TREATMENT

**Venous Pulsation.**—To understand this we must first glance at the anatomical and physiological conditions. The internal jugular vein has valves at its entrance into the subclavian, or these may be as much as three-quarters of an inch above this. The external jugular has valves both at its entrance into the subclavian and an inch-and-a-half above the clavicle. The subclavian has valves at a point just distal to the entrance of the external jugular. All these valves are only doubtfully competent and sometimes are absent. There is a constant pressure from the distal veins towards the heart. Owing to the resiliency of the lungs and the action of the heart, the blood is attracted from the veins to the chest ; this attraction is essentially a varying one, being more in inspiration and diastole than in expiration and systole ; but, though variable, it is always acting—except, perhaps, in extreme expiration with cardiac systole. Both this *vis a fronte* as well as the *vis a tergo* from the distal veins will act more strongly on the blood in the jugular veins of the right side than on those of the left, as the left innominate forms a considerable angle with the superior vena cava, whereas the right is nearly in a straight line with it. Gravity will aid both these forces in the erect position. Where the jugulars enter the subclavian they have firm fascial attachments which prevent their collapse at these points ; further, the internal jugulars are dilated near their openings into what is called the jugular bulb.



Any slackening in the *vis a tergo* will tend to empty the vein, any slackening in the *vis a fronte* to fill it. In anæmia there is a constant lessening of the *vis a tergo*, with a corresponding constant comparative emptiness of the vein. The vein is in no way distended, the slightest force will thus increase its diameter. Hence, if there is even a small slackening in the *vis a fronte*, the blood will at once lessen the rate at which it flows through the vein, and the vein will fill up a bit, since the blood continues to flow into it as before. This filling up of the vein shows itself to the eye as a pulsation.

*The Auricular Systole* produces such a slackening in the flow, and therefore the pulsation should be pre-systolic; and Drs. Ringer and Sainsbury, in a recent paper, have stated that this is a diagnostic feature between venous and arterial pulsation in the neck, as well as between this form of venous pulsation and that due to tricuspid regurgitation. This may be so; but it is a most difficult difference to estimate. The veins are so flaccid that the pulsation must be considerably delayed in its transmission; whereas the arterial impulse in the corresponding artery is almost simultaneous with the ventricular systole. It is thus very hard to say whether the pulsation is with the auricular or ventricular systole. But whatever the exact time of the pulsation itself, its cause is no doubt the auricular systole.\*

\* Since this lecture was delivered, Dr. James McKenzie has published a very able and exhaustive paper on Venous Pulsation, in the *Journal of Pathology and Bacteriology* for May 1892. In it he

Why does not this auricular slackening of the *vis a fronte* produce pulsation in health? Occasionally it does do so towards the end of expiration, or with unusually deep respiration when the slackening is excessive; but this, in my experience, is very rare. At the end of the superior vena cava, as it opens into the auricle, are circular bands of striped muscle extending up the vein for two-thirds of an inch; these must act as sphincters during the auricular systole. We have seen in anæmia how it is probable that the systolic contraction of the mitral orifice fails to take place; the same failure probably occurs here, hence some blood actually regurgitates back up the vein during the auricular systole, and hence the much greater frequency of the pulsation in anæmia, as the slackening thus caused must be considerable.

The pulsation is increased on lying down, or is only noticeable during recumbency. The reason of this is simple: in the erect position gravity, to a great extent, nullifies any slackening of the stream from a diminished *vis a fronte*—gravity is always acting, it is a continuous force—the *vis a tergo* is also a continuous force. We have, then, in the erect position a combined *vis a tergo*

goes far to prove, by means of tracings, that this pulsation is usually triple; that the first and smallest rise occurs during the end of the ventricular systole and, he believes, is due to the still contracting ventricle forcing blood into the now refilled auricle; that the second and larger occurs during the auricular systole, and is caused by the backward flow of some of the auricular blood into the superior vena cava; that the third and largest is a percussion wave due to the systole in the adjacent carotid; and that following this is a large depression in the tracing, brought about by the negative pressure of the now flaccid and dilated auricle.

—the pressure in the distal veins *plus* gravity—that is, the *vis a tergo* is a larger force in the erect than in the supine posture ; but the *vis a fronte* remains the same in amount. It bears, therefore, a smaller proportion to the total force moving the blood than in the supine position ; therefore any variations in its amount will have less influence upon the blood-flow, and will therefore be less liable to produce pulsations.\*

**The Bruit de Diable.**—If pressure be made with the stethoscope in the interclavicular fossa or over the course of the internal jugular vein on the right side, often (40 per cent. Landois) a continuous murmur or hum will be heard even in healthy people. In anæmia this same noise is heard when the stethoscope is placed on as lightly as possible ; very often on both sides of the neck, and sometimes over the external jugulars also. It is far more commonly heard in chlorosis than in other anæmic states, and the ease with which it is produced does not seem to depend entirely on the severity of the disease.

This murmur differs from all others in persisting continuously during diastole and systole without a break ; but this continuity of sound, though very characteristic, is not always present ; sometimes it is heard only during the diastole of the heart, or with inspiration and not expiration, or when the patient's body is erect and not when it is recumbent, or perhaps only after physical exertion. Again, it may be a

\* In anæmia there also occurs the pulsation due to functional regurgitation through the tricuspid.

distinct see-saw murmur during recumbency, and only become continuous during the erect posture. But even when occurring with diastole alone it is not hard of diagnosis, for with a little patience it will be found to vary considerably, and at times to be audible throughout both systole and diastole. In fact there is nothing to confound it with; the murmur of aortic regurgitation is so entirely distinct in quality, and its area of audition is so different, whilst the double murmurs of aortic valvular disease and aneurysm are markedly see-saw and never continuous.

As to its cause, Dr. Ogier Ward, of this city, so long ago as 1837 showed that it arose in the veins, and not in the arteries. Its exact causation is even now uncertain; its frequent occurrence tempts one to believe in its being due to some change in the nature of the blood; but that it cannot be due to any molecular variation in this fluid—which would increase friction, for example—is evident from its absence in the arteries, where the increased rapidity of the flow and greater pressure should make it far more prominent in these than in the veins. On the other hand, it cannot be due solely to any local cause, as it is heard (rarely, it is true) in the facial and crural veins, and with moderate frequency in the superior cava, subclavian, thyroid, and axillary.

The usual explanation given of its origin is that it is due to passage of the blood-stream from a narrower to a broader part of a vein, with the consequent production of a fluid vein. But there is one condition of the blood in anæmics which may favour the production of

this sound—that is, its diminished quantity. In health, with a normal amount of blood, there is an average negative pressure of 0·1 millimetre of mercury in the internal jugular, according to H. Jacobson; hence there will be a tendency to collapse of the vein walls where they are not filled with blood. In anæmia, then, with its smaller amount of blood, the veins will be less full; and therefore there will be more likelihood of collapse, or partial collapse, occurring. And it is this collapse, or partial collapse, which produces the *bruit*. It at once gives rise to a condition favourable to the origin of a fluid vein, the collapsed portion acting as a constriction. In reality it *is* a constriction caused by the external pressure of the atmosphere, this being greater than the pressure of the blood within the vein by the force represented by 0·1 mm. of mercury, and in anæmia probably more than this. The veins are never fully distended, except locally in pathological conditions, for they alone can hold more than twice as much blood as there is in the body. Any increase in the amount of blood will, therefore, more fully distend them and increase the pressure within them, and lessen their liability to collapse, and *vice versa* with a diminution of the amount of blood. Such diminution occurs in anæmia; hence the liability to collapse will be increased in anæmia. This tendency will also be increased by the poor tone of anæmic vessels, and their tendency to fatty degeneration.

We should not expect to obtain this *bruit* in the intra-thoracic veins; for though the negative pressure



within these is, if anything, greater, yet it is the still greater reduction in the external pressure, owing to the aspiration of the lungs and the systoles of the right heart, which produces this increase; hence one would expect the pressure inside the intra-thoracic veins to be always greater than that immediately outside them. Nor should we expect to find it so often in the veins of the left side of the neck as in those of the right, for the blood has to turn a double angle when coming from the left, and we know that this would greatly impede its flow, and therefore increase the internal pressure by damming up the blood. Again, on the right side it would occur much seldomer in the external jugular for a similar reason. In the subclavians we have one angle to be turned, and they are also partly subject to the intra-thoracic aspiration; hence we should not expect it so often in these. As we get farther from the heart the internal pressure becomes steadily in excess of that of the atmosphere, and so we should not expect collapse or its resulting *bruit*. The right internal jugular vein is therefore the one in which, apart from any local conditions, we should *par excellence* expect this *bruit*. And this is what we find clinically.

But there is also a constantly acting local condition where this vein opens into the subclavian; it forms a dilatation called the bulb, which is prevented from collapsing by its close attachment to the surrounding fascia. It is easy to understand that any small collapse of the vein distal to this will induce the *bruit*. The external jugulars have similar fixations at the points

where they debouch, and hence, no doubt, one chief cause of the *bruits* in these.

Any pressure on the vein will also produce it, as, for example, the stethoscope or some permanent constriction resulting from inflammation—as occurred in a gentleman under my care who had had a blow on the neck just behind the jaw, and in whom there is now an extremely noisy *bruit de diable*.

I mentioned that when in the erect posture a murmur was continuous it sometimes became a double murmur with recumbency. The first and usually the most prominent portion of this double murmur is found to be systolic, and the other diastolic, the time between the systolic and diastolic murmur being somewhat less than that between the diastolic and systolic. It is accounted for in this way : The quicker the flow, the louder and more constant the sound ; in recumbency gravity ceases to act, and therefore the murmur is less notable. Its peculiar rhythm is thus explained : Its systolic portion is occasioned by the rapid rush of blood into the empty right auricle whilst the right ventricle is in systole ; but this rapid rush ceases before the ventricular systole is over, hence a slackening of the stream occurs, for none can pass into the still contracting ventricle ; hence a cessation of the murmur. The ventricle, however, almost immediately relaxes, and there is a second rush of blood, lasting up to the auricular systole, with thus a diastolic *bruit*. It is therefore easy to understand the variability in this *bruit*, from a continuous hum of almost equal intensity to a regular see-saw.

**Treatment.**—Here we must remember that the affections of the vascular system are always of a secondary nature. It is the primary lesion we have chiefly to treat: what is, then, this primary lesion? Anæmic debility is always the result of some kind of excessive strain—*e.g.*, Puberty, Acute Illness, Poor Food, Dyspepsia, Anxiety. The fundamental want, as we have seen, is power to properly assimilate nourishment in sufficient quantity for the bodily requirements. It is proteid nourishment which chiefly fails, and this we found to be taken up and metabolised to a large extent by the mucous membrane of the alimentary tract. It is, therefore, an atonicity of this membrane which lies at the bottom of anæmic debility and all its secondary troubles, and our great aim must be to increase the energy with which this performs its important duties. But while we thus endeavour to improve the energetic income of our patient we shall also act most wisely if we diminish, as far as possible, the expenditure of such energy as he already enjoys, so that it may not all vanish in the current expenditure of his daily needs, but some may be retained to strengthen his constitution, may, in fact, go to the increase of his physical capital. Bearing in mind these two main lines we may consider treatment under the following heads:—

(1) **Climate.**—The three climatic requisites are warmth, sunshine, absence of strong winds: the first lessens the need for metabolism; the second cheers the nervous system and quickens life—that is, it increases the metabolic power; the third renders the muscular

effort of outdoor life much easier and outdoor existence generally pleasanter. Such a climate enables the invalid to spend the greater portion of his waking hours out of doors, idly sauntering, sitting, or driving ; and this is a most essential part of the treatment of anæmic debility, as the patient can breathe pure air and so stimulate all the oxidising processes of his body to the utmost extent, and make the best use of the food he takes by the mouth.

A seaside residence, such as Hastings, Bournemouth, or Torquay is most suitable for the whole year round ; if for the summer only, the more bracing climate of Margate, Cromer, and Scarborough, or the moorlands of Yorkshire may be chosen. A mountain climate is to be avoided in this as in all functional affections of the vascular system, especially if the constitution be erethic, as the rarefied air of high altitudes has a most disturbing influence on cardiac action. When erethism is well-marked, long sea-voyages or the luxurious air of the south-west of England will prove of great service.

(2) **Mode of Life.**—*Dolce far niente* should be the motto of him who suffers from anæmic debility ; to eat lotuses in a sunny clime his greatest aim. He should go to bed with the sun and so avoid the impurities of artificial light ; but by no means should he rise with it, but rather wait till the world is well awake and revived with warmth. The rest in bed should be of long duration, never less than eight to ten hours, and in severe cases this should extend to twelve, a light breakfast being taken before rising. A mid-day siesta—even



with men of business—should always be taken after lunch, if only for half an hour. No long sustained exertion, mental or physical, should be allowed, the sense of fatigue ever being looked on as the criterion of labour, and drowsiness always given way to. But more important still is the avoidance of any *sudden* toil and excitement. With the unstable vascular system of anæmia this is a very hard thing to do, but the patient should be carefully and persistently instructed to keep his nervous system constantly warned against the surprise of a suddenly called-for physical exertion, or the even more difficult thing to be guarded against—a sudden annoyance. Of a debilitated patient is the saying especially true—forewarned is forearmed. Worry and anxiety must be constantly tabooed. I say *constantly*, for it is wonderful how we can lessen or annihilate the petty worries and anxieties of life by constantly keeping one's mind set on having nothing to do with them. It is very seldom, even in health, that they are worth giving way to ; when we do attend to them is not our summing up afterwards nearly always *Le jeu n'en valait pas la chandelle*. To every patient with a functionally disordered heart, it is our duty to say—and in so saying to speak with the strongest emphasis we are capable of—On no account let small worries take you by surprise ; be always on your guard against them ; keep your mind in this fixed attitude—“ I will let nothing disturb the serenity of my life.” Let us insist that trains need never be caught, chances of making money must calmly be suffered to slip by, cross words and slighting sneers



must pass unheeded if contrary action should quicken the heart one iota of a beat beyond that which is pleasurable.

(3) **Occupation.**—Unless the constitution be thoroughly broken down by overstrain, or the anæmia be extreme, complete idleness is unwise. But from what I have just said it is evident that exacting employment must be renounced at any cost; its continuance can only result in increased debility. The work should be out of doors, involving no heavy toil; on this point, Tucker-Wise has made the valuable observation that of the anæmic girls who came up to the great hotel on the Maloja in the Engadine, those who had to work indoors as cooks, kitchenmaids, &c., got no benefit, whilst the nursemaids who took the children out every day soon markedly improved. A middle-aged patient accustomed to a sedentary life must be very chary of undertaking outdoor employment, even light labour will be to his muscular system a toil, and it will often be better for him to continue at his sedentary work, especially if he be able to live in a climate mild enough to have the windows perennially open. The agent of a small estate, or the manager of a small market-garden seems to me the most suitable kind of occupation. But in most cases rest and change for six and twelve months is the thing needed, and will prove sufficient for the restoration of health. A summer spent on the Lake of Geneva with the succeeding winter along the Riviera would answer well.

(4) **Food.**—The nature and amount of this will

depend upon the condition of the digestive organs. As a rule it should be given frequently (six meals a day) in small amounts, and of plain but nutritious character, containing plenty of proteids. I think this high proportion of proteid food should also be given in pernicious anæmia, believing, as I do, that it also is a disease of defective hæmogenesis; but if you consider it one of increased hæmolysis, to be logical you must limit your proteids, as Dr. Stephen Mackenzie has pointed out, since meat diet increases the activity of the destruction of the red corpuscles.

In the debility of middle age, forced feeding is often of very great value. To carry this out successfully the patient must be confined to bed and oxidation must be kept up by massage; the massage being further of great assistance in removing neuralgic pains, which often in these cases seriously detract from the cure of the patient. In October 1886 I saw in consultation with Messrs. Clifton and Bremner, of Leicester, a lady, whose præcordium was so tender that she shrank from the very lightest percussion and even palpation; there was further much pain down the left arm, especially in the wrist and hand. After a month of massage, this pain was quite gone, her appetite greatly improved, and her previous nervous prostration changed for an attitude of bright hopefulness.

It is wise to limit the ingestion of liquids as the greater the bulk of the blood the harder the work of the heart; moreover, the blood is anæmic, so that lessening its quantity should improve its quality.

(5) **Drugs.**—In chlorosis, iron is on all hands admitted to be *the* drug. It is very rarely that I do not at once begin with it, except in cases of severe gastric disturbance. When the appetite is wanting, and the stomach sensitive with atonic constipation, I order a tonic of nux vomica and soda with peppermint water and tincture of hop, adding to it, if there be much nervous upset, 10 to 15 drops of bromidia. This I give shortly before each meal, and after the meal this pill—aloes socotrinæ, gr. j; extr. belladonnæ, gr.  $\frac{1}{4}$ ; ferri sulph., gr. j; conf. rosæ, q.s.; giving sufficient of these—one to six daily—to keep the bowels well open. So soon as the digestion has improved I add Blaud's pills, one to six a day, and, when the appetite becomes good, cease to prescribe the mixture. You perceive that I adhere to the old-fashioned form of iron which is contained in Blaud's famous pill. Very rare indeed are the cases of chlorosis where I have found it ill borne; then I have met with fair success with Denaeyer's peptonate of iron or liq. ferri dialysati, which latter I remember Dr. Wade speaking well of for cases which could not stand the cruder forms. The theoretical advantage of Blaud's pill is that in it you prescribe ferrous carbonate in a nascent condition; but the Addendum to the *Pharmacopœia* 1890, and Mr. Martindale also, states that whereas a 5-grain pill should contain a grain of ferrous carbonate, yet before it can be dried and coated, quite half of this is converted into ferric oxide. If this be so, probably before the uncoated pill is swallowed, very little carbonate remains

at all ; hence the desirability of coating them, and more especially of the bi-palatinoid disc which prevents the two ingredients mixing till they reach the stomach, and should, therefore, give the full proportion of nascent ferrous carbonate. In private I prescribe these bi-palatinoids, but for hospital practice they are too expensive. Dr. Mackenzie uses ferri sulph. exsiccatum with the carbonate of potassium, but Mr. Campbell, our able chemist at the Queen's Hospital, tells me that this salt is nearly always overheated in the process of drying, and an insoluble basic sulphate to some extent produced, which evidently cannot be converted into ferrous carbonate. Whatever form of iron be employed, it must be given in large amounts ; many chlorotics will take 30 or more grains of Blaud's pill daily, and will do much better on this than any less quantity ; the iron should be continued, too, for two or three months after the patient feels quite well, and has recovered her proper colour.\*

In other forms of anæmic debility iron ceases to have this apparently specific action. Its value is still undisputed, but it is only one of several other valuable agents, and in my experience by no means the most important. In the debility of middle age and exhaustion from overstrain, it is, as a rule, but badly borne,

\* I believe there is a useful future before the proteid forms of iron—hæmoferrum, hæmol, hæmogallol, ferratin. I have found them excellently borne by severe cases, and in one post mortem the stomach contained several pills in process of dissolution, but scarcely a trace of iron (to the eye) in the gut beyond (December 1894).

while strychnine with hydrochloric acid has far greater remedial effects. Pernicious anæmia stands rather by itself in this relation ; it and chlorosis are at the two extremes of the ferruginous scale, iron having less influence over this than over any other form of anæmia. Yet if pernicious anæmia were due to the increased destruction of red corpuscles, and the consequent increased elimination of iron as a waste product from the system, surely it is here where an increased supply of iron should be particularly efficacious as, the hæmogenetic functions of the body not being interfered with, the more iron they had the more hæmoglobin they would be able to make, so as to compensate for its increased destruction. But if the excess of waste iron be as I have suggested, due to inability of the liver to make use of it, then it is evident that an increased supply would not prove of much benefit. First of all we must improve the iron-distributing function of the liver, and then we can hope to benefit from the administration of iron. This is what we have already found to be the best method of treatment clinically. In pernicious anæmia arsenic is recognised as our most valuable drug, especially when borne in large doses (10 to 15 minims). Now arsenic lessens the glycogenic function of the liver, and it also hinders the crystallisation of hæmoglobin—that is, it influences in an important way the properties of both the organ and the substance which are in question. It is therefore not mere vague theorising to believe that it may aid the liver in hæmogenetic labour.



We also find that the recovery of patients suffering from pernicious anæmia is hastened by the administration of iron. This, also, is what we should expect on my theory. When once the liver has been improved by arsenic, so that it can again perform its hæmogenetic function, then the case becomes one of ordinary anæmia, and iron acts as it does on this latter disease. There is now much evidence and authority in favour of the statement that pernicious anæmia is not necessarily fatal. I saw in 1891, in consultation with Dr. Haig of Coventry, an old gentleman whom I was told Dr. Broadbent had previously diagnosed as suffering from pernicious anæmia. You will agree with me that no physician is to be more trusted in such a matter than Dr. Broadbent ; yet when Dr. Haig and I examined the blood, it was normal as far as the corpuscles were concerned ; and I heard some months later that the patient was much improved in general health, and quite cured of the general eczema for which I saw him.

In men, both young and middle aged, the vascular symptoms are often prominent, and produce feebleness and languor apart from any digestive trouble. In these cases nothing in my experience answers so well as the prolonged use of digitalis,\* the amount varying with the individual from 10 to 20 or even 30 minims of the tincture taken thrice daily after meals. A sedative,

\* I have for the last three years nearly always prescribed Nativelle's digitalin, ordering one granule once, or perhaps twice, a day. This method is very easy of exhibition, and I find busy men will more steadily persevere with it than with a mixture.

such as a glass of malt liquor or of whisky taken daily with the dinner, is frequently of great service, or a drachm of the tincture of hop or twenty drops of bromidia may be added to the mixture. Sluggish bowels must be kept well opened by the pill I have spoken of when dealing with chlorosis and, when the digestive functions will stand it, Blaud's pill may be administered. If iron cannot be borne I have seen most excellent results obtained by adding quinine to the strychnine. The following are examples of the prescriptions I should advise so far as the mere drug treatment of anæmic debility is concerned :

R Træ. digitalis,  
Træ. nucis vomicæ,  
Acid. hydrochlorici, āā ℥x;  
Aquam ad ʒj.

Sig. To be taken three times daily after meals.

R Pulv. digitalis,  
Quininæ sulphatis, āā gr. j;  
Extr. nucis vomicæ, gr.  $\frac{1}{2}$ ;  
Exc. q.s. ut fiat pilula una.

Sig. To be taken thrice daily after meals.

R Liquoris strychninæ, ℥v;  
Sodæ bicarb. gr. v;  
Træ. lupuli, ʒj;  
Aquam, ad ʒj.

Sig. To be taken thrice daily before meals.

Further, I would not have you forget the efficacy of mere local mechanical treatment for the strengthening of the heart when this organ is distressing the patient with its dilatation and palpitation. Strips of belladonna plaister an inch-and-a-half wide, of sufficient length to

reach from sternum to spine, and so applied that they cross one another at an acute angle, the point of crossing being situated over the apex beat, are often a great help in quieting the heart's action. Six to eight of such strips should be applied just as one straps the chest for the pain of acute pleurisy. They should be renewed every three or four weeks, and persevered with for six or eight months.

Finally, let me advise you as to your method of procedure in prescribing for this class of patient. I usually give them three papers, one dictating their mode of life, another the arrangement, &c., of their food, and a third for the chemist. You will find them sadly lacking in the energy of perseverance; you must act the schoolmaster to them, and leave nothing to their initiation; they are too weary to follow up hints or to translate into detail a general instruction; but detailed commands they obey implicitly. Moreover, this particularising of their life enables you speedily, at their next visit, to detect any deviation from your previous prescription, and to at once bring them to book concerning it.

My opportunity is now over. To me the labour of working out these lectures has been both a pleasure and an instruction. I can only hope that to you the delivery of them has not proved too irksome, and that you have reaped some little knowledge as a reward for your patient listening, for which I thank you most heartily.

## VIII. ARTERIAL HIGH TENSION

ARTERIAL high tension may be divided into three kinds : cardiac, degenerative and vasomotor. **Cardiac** high tension is that variety which results from an abnormal contracting force in the left ventricle, either owing to some temporary disturbance—as excitement, exertion—or to some permanent cardiac defect, which prevents this force being used so economically as usual—as in aortic regurgitation. **Degenerative** high tension is that resulting from a degeneration of the arteries, which makes their walls thicker and more rigid ; therefore, it requires greater force to drive the normal amount of blood through them than would be the case with healthy vessels having normally expansive walls. **Vasomotor** high tension is brought about by increase in the the contracting force of the peripheral arterioles, which thus, as in the degenerative form, necessitates more propelling power to pass a given quantity of blood through them than would be required by normally contracting vessels. It is evident that there is one condition necessary for the production of any form of high tension ; there must be extra contracting force put forth by the left ventricle. Now, excessive use of any portion

of the body is to be avoided, more especially of such an organ as the heart, which knows no rest. In so far, therefore, high tension is always objectionable, and unless there are counterbalancing reasons of great moment, it should be our endeavour always to do away with it. Speaking generally, there *are* such reasons in the forms I have called cardiac and degenerative, but not in the vasomotor variety. In aortic regurgitation, which we may take as the type of the cardiac form, some of the blood forced into the aorta flows back into the ventricle ; if, then, say, three ounces be the amount the individual requires to be sent into his arterial system at each heart stroke, when regurgitation exists a larger amount—say, four ounces—will have to be similarly propelled to allow of the reflux into the ventricle of one ounce. Four ounces will distend the aorta more than three, and the more an elastic tube is distended the greater is the tension of its walls—that is, the tension in the aorta will be increased, and therefore there will be high tension throughout the arterial system. But as there is no excess of resistance this high tension will force the blood through the arteries at a greater rate, so that the increase in fulness of the arteries will be of shorter duration than normal—that is, the pulse wave will be short, and the period of emptiness between the waves will be long. This is why the aortic regurgitant pulse is called the pulse of unfilled arteries—a condition due to the force of the heart being greater than is required by the peripheral resistance. So that, apart from the regurgitation of blood, the aortic pulse would



still be one of high tension of short duration, followed by an unusually long period of very low tension in the arteries, the reflux of blood merely intensifying this latter condition. The high tension which occurs with the increased cardiac action during muscular exertion is of the same nature ; but here the period of low tension—that is, the collapse of the pulse—is not so marked, as there is no reflux of blood into the ventricle. In the healthy subject this form of high tension is very transient, as Nature quickly provides a corrective by making the peripheral resistance subnormal, and so allowing the same cardiac force to keep up a more rapid circulation. Similar to this is the high tension due to palpitation, but the condition is a more serious one, and its mode of ending uncertain. We are dealing with a pathological state, and not a physiological one, as in the high tension of exercise. It is unlikely that the peripheral system will accommodate itself to the new condition and diminish its resistance ; very probably the same causes which induced the palpitation will also have increased the peripheral resistance. Hence the heart fails to empty itself during its shortened systole against the increased pressure, and dyspnœa, oppression, or even faintness follows.

It is given to but few mortals to die altogether—from a universal failure of function. Unfortunately for man, as a chain's strength is that of its weakest link, so is the strength of his life dependent upon his weakest function. Death comes to most men through the failure of one function. To some it happens, either through inherit-

ance or the character of the life led, that the arterial system is the first to decay ; arteries thicken with the rigidity of fibrous tissue or calcareous deposit, and their gentle, ever-varying elasticity is a thing of the past. The heart has to force the blood through a system of comparatively unyielding tubes, and we know that this requires greater exertion. Hence, with too quickly decaying arteries, the blood tension must be increased if a normal circulation is to be maintained. Health in its true sense is out of the question ; at best there is but a pathological equilibrium. One of the hardest things a physician can have to determine is what tension is requisite for this. If he allow it to remain too high, cerebral hæmorrhage stares him in the face ; while if it remain too low, then symmetrical gangrene, benumbed extremities, and all the woes of vascular debility make wretched the life of the patient. The character of this degenerative tension differs greatly from that which is primarily due to cardiac action. It is no question of forcing a larger amount of blood through pleasantly accommodating tubes, but one of pressing the same amount through tubes which will not enlarge. This necessitates that the tension throughout the *whole* of the period occupied by the passage of blood must be increased. There will be no period of subnormal or even normal tension. Moreover, as the system is rigid, the increase of tension conveyed by the ventricle to the aorta will be felt almost simultaneously throughout the system ; hence the onset of tension will be sudden, as in the high tension due to cardiac action.

In high tension due to increased contraction of the arterioles while their elasticity remains normal, we again find the need to force a normal amount of blood through abnormally small tubes. The tension wave will thus, as in the degenerative form, be a long one, persisting through the whole of the diastole ; but, as the tubes are not rigid, its onset will not be sudden. This increased contraction of the arterioles arises from undue excitement of the vasomotor centre owing to some morbid element in the blood circulating through the medulla. This morbid element may have a very transient existence, or may continuously act for several days, as is exemplified in the varying duration of megrim from a mere headache of an hour to an utterly prostrating bilious attack of a week. It may frequently recur throughout life, as in chronic renal disease, and so lead to the deposit of fibrous tissue in the arterial coat with its consequent thickening ; but even in chronic renal disease it is never always present. The tension may be constantly high, it is true, but this will be owing to the vascular degeneration ; and however high this mean tension may be, there will be exacerbations of it when impure blood excites unduly the vasomotor centre, just as megrim causes exacerbation of the normal tension ; and, unfortunately, there will be depressions of it below this mean whenever the heart flags and fails to be equal to its perennial increase of toil, as nearly always happens in the later stages of renal cirrhosis.

From what has been stated it is manifest that the treatment of high tension is not always one and the

same. Sometimes it is to be left as it is, sometimes it should be encouraged, and, perhaps oftenest, it is to be diminished. I say perhaps oftenest, not because it is necessarily this form which exists most commonly, but because it is the one which most frequently produces symptoms, and therefore is the one for which our aid will be oftenest sought. That we should most carefully differentiate these varieties need not be insisted upon, for the treatment required for one patient may be of an exactly opposite character to that required for another. The three forms I have described may be separated from one another by examination of the pulse. The pulse of cardiac high tension is felt beneath the finger as a wave of great size, but very short duration. It comes suddenly, strikes forcibly, and vanishes as quickly, leaving apparently nothing behind it to mark its passage. The wave of the degenerative pulse comes almost as suddenly, but is much smaller, and is so loth to go that much seems still to remain, even up to the time when its successor takes its place. The wave of vasomotor high tension feels smaller than either of the others and, unlike them, comes laboriously and slow. Its duration is long, but it appears to have vanished, or nearly so, before its successor arrives. So long as only one of these forms exists and remains quite distinct its diagnosis is easy; but generally two, or even the three, are all acting as components in the resultant high tension. Then the apportioning its correct value to each component is often a most delicate and difficult task, but one which it is of great importance to accurately

perform. I always set about it in this way. First, I attend only to the shock of the impulse—is it large, small ; compressible, incompressible ; sudden, or slow-coming ? This will tell me the condition of the cardiac action. Next, I feel for the state of the pulse after this shock is over—does it remain full, or nearly so, or empty slowly, or vanish quickly ? Does the tube feel large or small, compressible or incompressible ? and so I learn whether the peripheral resistance, the vaso-motor contraction, is great or slight. Finally, I compress the artery, both distally and centrally, with my index and ring fingers and, with my middle finger, still feel for the comparatively empty vessel.\* Can it be felt, and, if so, how plainly ? Is it nearly as evident as before, rounded and firm, or are its walls soft and flaccid ? If it stand out firmly, does it feel tough, like a round leather bootlace, or ribbed, like whipcord ? Then, having made these three distinct observations, I compare them together, and estimate how far one should correct the conclusion drawn from another. For instance, if I find the vessel is degenerated, then the

\* In the case of the radial this distal compression is important, as a recurrent pulse, both with high and low tension, is not rare ; and, if this do not exist, the back-flow is usually sufficient to moderately distend the vessel. If I am still doubtful, I isolate the vessel with the thumb and the fingers of the other hand. I thus have two or three fingers with which to feel the vessel, and plenty of space at my disposal. Moreover, one's fingers—if one be not a musician—are so wont to work together that it is difficult to make the delicate varying movements necessary to estimate the state of the vessel with one of them whilst two others are producing firm, continuous pressure. In practice I frequently make use of both hands.



incompressibility and suddenness of the impulse will have their significance lessened; and its smallness, if such exist, need not mean that the ventricle is acting feebly. Again, should the impulse be slow in coming and small, a sluggish and feeble circulation is indicated if the tension be normal or subnormal; but should this be high, then this slowness of onset is owing to the extra fight the heart has to make against the tightly distended aorta, and the length of its duration will make up for its smallness.

What I have more to say will be told best in connection with some illustrative cases, to the relation of which I will now address myself.

A widow, aged 57, of apparently robust physique, came to my out-patient clinic at the Queen's Hospital last October complaining of a stiff neck, an indefinite but painful swelling below the left ear, cramps in the calves, sleep disturbed, flatulence, alternate shiverings and flushings. Her tongue was thinly coated with a white fur, but her bowels were regular. Her aortic second sound was much accented, the tension in her radial was high, and the impulse was forcible. All her symptoms were no doubt due to a gastro-intestinal catarrh probably resulting from a chill which, interfering with assimilation, had allowed morbid elements to circulate in the blood, some of which had irritated the vasomotor centre, and so produced high tension. Here was a simple, uncomplicated case of vasomotor high tension, and treatment must be directed to the removal of the morbidity in the blood which had induced this. We must be careful not to look upon the high tension itself as the evil; it is, as we know, a preservative measure, to protect the tissues from the impure blood. But we naturally conclude that if we lower the tension we shall have removed the

impurities in the blood which raised it. It is true that the malassimilation of gastric catarrh may have produced impurities other than those which raise tension, but they all appeared together, and we act in the hope that the drug which is capable of removing some will be efficacious in eliminating all. Now mercury, especially as the subchloride, is the drug *par excellence* for correcting high tension due to digestive derangement. It does not do this by virtue of its aperient action, for it is a slow-acting aperient, while a single dose will often do away with high tension within two hours. Moreover, a small amount—a quarter of a grain—may be quite sufficient to lower tension, though entirely inadequate to open the bowels. I therefore gave this woman an eighth of a grain of calomel twice a day. She came to me again in a week with all her symptoms gone, or nearly so ; practically well. Her tongue was clean and moist, and the tension in her radial decidedly less, her pulse being correspondingly shorter and more sudden.

Mr. —, aged 50, a well-to-do tradesman, came to me in February 1887 complaining of sleeplessness, from which he had suffered for three years—ever since he had lost his partner in the business ; also of dyspnœa on even slight exertion, of occasional giddiness and swooning, and of much diffidence and depression. His history was very good, except that he had had two very slight rheumatic attacks, lasting two or three days each, four years ago, and that from a child he was always nervous, the least unusual thing making him turn pale—*i.e.*, his cardiac government was unstable. His heart I found increased in size, with a diffused pulsation in the fifth and sixth spaces near the sternum. The sounds were slapping in character, and the second aortic was unduly accented. His arteries were thickened, and his pulse was 80 ; irregular, sudden and incompressible. Here the condition was a complicated one. He had degenerated vessels. These I could not alter. His tension was high, but whether too high, considering the

difficulty there must be in passing a due amount of blood through his diseased vessels, I could not determine at a first visit. But there was one condition I knew wanted something done for it—his heart was failing. With high tension his pulse should have been slow, but it beat 80 times in the minute; moreover, it was irregular both in size and rhythm; his heart was increased in size, and this increase was probably due to dilatation, as the sounds were not those of hypertrophy, and there was a diffused impulse over the right ventricle. I therefore prescribed the following heart tonic three times a day:—One drachm of the infusion of digitalis, one grain of nitrite of sodium, five minims of liquor strychninæ, five grains of bicarbonate of soda, compound gentian infusion to one ounce. I added the nitrite of sodium to counteract the action of the digitalis in raising vasomotor tension, as I was not certain that this might not already be too high. I also gave him a drachm of paraldehyde to take at night should sleep refuse to come to him. He came again in a fortnight feeling much better, with a franker, brighter expression. His pulse was 67, not incompressible, of fair size and regular, except that it had eight intermissions in the minute, which were represented by abortive heart-beats. The heart sounds were no longer slapping, but the second aortic was still increased. During eleven of the fourteen nights he had slept six hours without the paraldehyde, and on the other three he had slept four hours with it. He was ordered to continue the same treatment, with the exception of the digitalis, which I withdrew, thinking it might account for the intermissions. Fourteen days later I saw him once more. His expression was now quite frank and open. His face had filled out, and his complexion become ruddy. He felt quite equal to his work, and took pleasure in it. He had had six hours' sleep on twelve of the nights. His aortic second sound was no longer increased; the pulse was 80, with four intermissions, but the abortive heart-beats corresponding to these were stronger than formerly. I was rather surprised at

this result ; with his thickened arteries I should have expected his machinery to work best with a tension above normal, but it is evident that a normal tension was most suited to him. Still, this normal tension could not have allowed him to have a normal circulation ; this must have been an enfeebled one and, consequently, he could never be equal to the work his constitution might have performed if there had been no thickened vessels, nor to that work he might have done if his heart had been strong enough to cope with thickened vessels. What happened was this : he found his circulation was most active when his tension was normal ; if it rose above normal his heart failed, and if it became subnormal the thickened vessels too greatly cut off the supply.

It is in such cases as these that the medical attendant who has experience of his patient has such an immense advantage over the chance consultant. If I see this man again, I shall bear in mind that he works most easily with a tension which is the normal tension of the healthy individual.

Mr. —, aged 36, consulted me in May 1891, complaining of a chronic cough, with thick mucoid expectoration and attacks of præcordial oppression and faintness. Three years ago he had congestion of the lungs and, a week before I saw him, spat up a teaspoonful of bright blood just after he had assisted in getting a boat over a weir. There was a little crepitation at the base of the right lung, but no sign of phthisis. His heart's apex beat in the sixth space and left nipple line, the area of dulness being otherwise normal. The impulse was excited, but the first sound was fairly long and firm. His radial was thickened. Whilst he was standing up for me to examine his chest he was seized with one of these attacks of oppression, his heart's action becoming slow and laboured.



He was quite calm, refused any stimulant, but said he must lie down on the floor and then the attack would soon pass off. He did so, and I kept my fingers on his pulse and my stethoscope on his heart. I noted that as the symptoms passed away the heart beat more quickly and with less strenuousness, while the pulse became full and soft. He told me that though he occasionally got palpitation on exertion, yet by far its more frequent cause was indigestion. And so it seemed, for I told him at once to go and do what business he had in the City and then return to me. In about an hour he came back. He had been bustling about and walking quickly, yet his heart was acting much better than when he left me. This, then, was an instance where impure blood raised the peripheral resistance to such a degree that his somewhat feeble heart was unable to cope with it. This is a condition very common amongst sedentary folk and brain workers. Their vascular nerves become very sensitive, whilst the heart muscle is not of the strongest. As a result, reflex action is too easily excited, and the heart is too easily upset by the shock thus created. Especially are those people liable whose vessels are unduly rigid and who therefore already give their heart excessive work to do ; in such subjects a small increase in the vasomotor contraction may produce a result as great as a far larger increase in a subject with normal vessels. But to return to my patient. My prescription was:—1st. To lead an even, quiet life, with as much fresh air as possible, but no severe exercise of any kind. 2nd. Rhubarb powder, bicarbonate of soda, ginger powder, two grains of each ; to be taken daily before dinner. 3rd. A quarter of a grain of subchloride of mercury to be taken twice or thrice a day during the dyspeptic attacks. 4th. Powdered Carlsbad Sprudel salt, a drachm to be taken before breakfast in hot water when required, and a tumblerful of hot water to be taken regularly night and morning. 5th. Five grains of antipyrin every half-hour for three doses during the attacks of palpitation. He lived some distance off, and I did not see him



again, but he kindly wrote, in answer to my inquiry, that he had received considerable benefit from the treatment, except from the antipyrin, a second dose of which several times made him feel faint.

This action of antipyrin is, I think, a thing to be mindful of. It is a drug we should very charily prescribe in high tension (megrim, for example), unless we are sure of the heart's stability. I believe that its depressing effect on the heart occurs sooner than its action on the vasomotor centre. But in every case where we use antipyrin we must remember it merely removes an effect, it does not touch the cause. Its action in depressing the vasomotor centre is stronger than that of the impure blood in stimulating this centre. Another drug, such as calomel or rhubarb, which will prevent the formation of the impurity, must be given as well.

The effect of high tension upon sleep is a very important one ; it is also a complex one, acting in different ways.

Mr. —, aged 56, suffering from syphilitic phthisis of long duration, was sent to me on account of persistent insomnia, from which he had suffered for nine months, ever since the death of his wife. He told me that his average amount of sleep was less rather than above two hours, that during the rest of the night his brain was incoherently, yet pertinaciously active, and he spent the dark hours in tossing restlessly to and fro. But in the day things were very different with him ; he was ever languid and drowsy, often dropping to sleep in his chair, and incapable of applying himself to business. His

heart's apex was ill felt in the nipple line and sixth space ; its dulness reached up to the third cartilage, and the second aortic sound was strongly accented. His pulse was 64, and though it appeared of fair size this was no doubt owing in a great measure to the thickened vessel, as the tension was very high. The urine was uratic. The explanation was simple. His grief had depressed his vital powers, including his digestive functions ; malassimilation and high tension resulted. During the day his feeble heart was unable sufficiently to cope with this ; on account of gravity the brain was the first to suffer from the consequent anæmia ; hence the drowsiness, &c., by day. But at night things were quite different : the heart had no longer to pump blood up to the brain or bring it up from the dependent legs, nor had it to spend its strength in exercise and the processes of digestion ; the impurity of the blood would also lessen every hour since the last ingestion of food ; hence the tension would relax. The brain would now be on a level with the rest of the body, and hence gravity would no longer injuriously affect it. For all these reasons its blood supply would be much greater than by day, and hence sleeplessness. He had taken in vain many soporifics, including opium, with no good result. He was already on a tonic regimen, so I merely prescribed a quarter of a grain of calomel to be taken three times a day in addition. In a week he came again. He had slept from eleven to four every night, and on two nights had dozed after four. His urine was clear, his pulse 68, and its tension very slightly above normal.

Sudden exposure to cold often seriously embarrasses the heart by the superficial high tension it causes. To come out of a warm house on a frosty morning and run to catch a train is more likely to produce palpitation and breathlessness than the same proceeding on a warm day in summer.

A fruiterer's assistant, with a double aortic murmur and a dilated aorta, came to me at the Queen's Hospital complaining of anginous attacks, which he said often occurred on his leaving breakfast to come out into the cold of the open shop to serve a customer, or when he drove out in his cart, if the wind blew coldly against him at starting. I gave him strychnine to strengthen his much broken-down heart, hydrochloric acid and hop to improve his digestion and soothe his nerves, with glonoin to take when his attacks came on. These attacks he had been having two or three times daily, and each lasted over an hour. During the next three months he only had four; and at the end of this time, though it was midwinter, he found himself able to face the cold with impunity. The great relief he got from the glonoin when he used it during the attacks showed that they were due to the injurious effect of suddenly occurring high tension upon an enfeebled heart; but when this organ was strengthened by the tonic regimen it was able to cope satisfactorily with this without such aid.

High tension may produce general œdema if the heart be weak.

J. R——, 33, was admitted into the Queen's Hospital under my care, on March 19, 1891, on account of orthopnœa and dropsy. He had double aortic disease; his heart was acting in a banging, troubled manner, though regular; its apex beat one inch without the left nipple line in the sixth space, and its dulness reached up to the second cartilage, though it was not increased to the right. His pulse was 100, collapsing; there was considerable tension between the impulses. Urine a cloud of albumen, but urea 2 to 2·8 per cent. On the 19th, the day of admission, he was given the following mixture three times a day: three minims of the tincture of strophanthus, one drachm of the spirit of nitrous ether, to one ounce of camphor water. On the 21st, when I first saw him, a grain of sodium

nitrite was added to this, and he was also given a third of a grain of calomel every two hours ; and on the 23rd the calomel was reduced to three doses a day. The effect on the urine, dropsy, and heart was remarkable. The urine passed during the first twenty-four hours after admission was 16 oz. ; on the 21st—*i.e.*, the day the calomel was first given—the amount was only 22 oz. ; but, on the succeeding days, 108, 118, 119, 97, 96, 64, 54 oz. respectively. On April 1st the œdema was quite gone, and also the dyspnœa. The pulse was much more collapsing, though less forcible, the tension between the impulses being subnormal, and the heart was acting with comparative quiet, and comfortably.

I think this result is instructive, for we usually raise tension to get rid of œdema. I should have done so in this case had it not been already high, and I feared the dangers of a too excessive tension on the damaged heart. I thought the heart was working up to its limit, and that any increase in tension would only weaken it and still further depress the circulation.

Many people complain of breathlessness after meals. This is usually put down to mechanical causes, such as flatulence, or a full stomach pressing up the diaphragm. But often there is no flatulence, nor evidence of overlaid stomach ; the subjects of it are not seldom most careful in their dietary. In several instances I have found it to be due to high tension, resulting, no doubt, from imperfect metabolism. Often, I expect, when flatulence *does* exist, high tension also plays an important part.

Grief kills ; but so, they say, does sudden joy. Joy quickens and stimulates cardiac action. “My heart leaps up when I behold A rainbow in the sky,” sang

Wordsworth. But suppose there is no field on which this joyful heart can display itself ; suppose the blood canals are blocked with high tension ! What then ? The bounding heart is unable to suffuse with joy the peripheries of the body ; its glowing warmth recoils discomfited upon itself. It cannot leap up. A great nervous revulsion occurs, and death comes. This is how it is that to some a great joy brings a ruddy glow and a feeling of the fulness of life, but to others deathlike pallor and a sense of dread oppression.



## IX. NOTE ON CONGESTION OF THE LUNGS

I WOULD like to add a short statement of my view of the pathological process of pneumonia, especially with regard to that strangely misleading misnomer Congestion of the Lungs.

When we hear of a patient having died of congestion of the lungs, what clinical signs and symptoms do we believe the medical attendant discovered? First, there was the negative condition; he could find no cause for death outside the lungs. Secondly, it was not bronchitis—that is, the disease was more or less localised; there were few, if any, rhonchi; there was little, if any, dyspnœic distress; the temperature was too high; the expectoration was too scanty for chronic bronchitis, nor was there evidence of successive attacks. Thirdly, there was no sign of consolidation—*i.e.*, no bronchial breathing nor marked dulness, nor increased vocal resonance; therefore it was not pneumonia. Now it is in this last conclusion that I think we err. The presence of these three signs is, no doubt, of extreme importance in telling us of the intensity of the pneumonia, but their absence by no means demonstrates its non-existence. We cannot too much impress upon ourselves the fact that they merely indicate an acoustic

condition of a lung—viz., that it is more solid and at the same time a better conductor of sound than normal.

As to the positive signs and symptoms detected, they would be these : more or less localised, medium sized, and fairly moist crepitations ; none, or very few, rhonchi, and these localised and in the small tubes (sibili) ; very little, if any, change in the percussion note ; no noticeable change in the quality of the breath sounds, but a distinct diminution in their quantity ; temperature of a bronchopneumonic type, and mild in degree.

By the right of what knowledge do we call this condition congestion of the lungs ?

Congestion, I take it, means an undue fulness of vessels, veins, arteries, capillaries, lymphatics, but not an exudation from them. Have we any evidence that such a condition, pure and simple, in the lungs gives rise to the above, or indeed to any, definite signs ? I think not. It is true that in those instances of extreme rarity when death has come in the first few hours of sthenic pneumonia such a condition has been discovered post mortem, and prior to death a slightly dulled percussion note with lessened breath sounds existed, but no crepitations. Crepitation necessarily means exudation ; and in all these cases of so-called congestion of the lungs there is exudation, but it is moderate in amount and in the intensity with which it is poured out. Hence there is no solidity producing dulness on percussion or increasing (resonating) the laryngeal

breath sound, or indeed so blocking the alveoli as to prevent crepitation and alveolar breath sound. The air-sacs are only partially full ; they move somewhat, though feebly, with respiration. Hence their contents (air and liquid) move on each other and against their walls (*i.e.*, crepitate), and the breath sounds are mixed (*i.e.*, vesicular) in character, but lessened in amount, as the difference between extreme expansion and contraction of the air-sac is less by the amount of the secretion it contains.

Pathologically, then, as well as clinically, congestion is a sluggish form of pneumonia. Congestion, catarrhal pneumonia, bronchopneumonia, lobular pneumonia, fibrinous pneumonia, and hæmorrhagic pneumonia are but the names of an ascending series of one and the same clinical and pathological condition—viz., that of active inflammatory exudation into the air-sacs of the lungs. They all have one essential and distinctive feature—that of active inflammation ; and to this feature we have given the name pneumonia. Let us then adhere to this title, and to this alone. It may be convenient to indicate sub-divisions ; if so, I think the terms hæmorrhagic, fibrinous, and catarrhal would be safe and sufficient ; or, indeed, as the examples of the first of these are very rare, we might speak of fibrinous and catarrhal pneumonia only. These terms are, I believe, clinically and pathologically co-extensive and co-equal. The more acute and sthenic the attack the more solid the lung, the more fibrinous the exudation ; the more sluggish the onset and the less the signs of

solidity, the more cellular (catarrhal) the exudation becomes. Further, severity of attack being equal, the more cellular the exudation the more dangerous the disease, and the less likelihood is there of its thoroughly clearing up.

I would ask you, then, to think seriously upon this question, and to see whether, as years make you richer in medical experience, your views do not agree with mine in this matter, and so resolve to have nothing to do with this phrase Congestion of the Lungs, thus simplifying and purifying the nomenclature of medicine ; for it is in your hands that this power and duty will lie.

## X. ON HÆMOPTYSIS

THE spitting of blood is a symptom of very wide range and great complexity. In my communication to the Society to-night I shall confine myself to that hæmoptysis where the source of the bleeding lies in the bronchi or parenchyma of the lungs, where the blood comes from the bronchial or pulmonary circulation. But even so, I fail to avoid the perhaps chiefest difficulty of all : to wit, the question of bronchial hæmorrhage.

It used to be thought that, because in cases of fatal hæmoptysis the bronchi were often widely and deeply stained with blood, the hæmorrhage arose within these tubes in most instances ; but more careful post-mortem examination, by showing the integrity of the bronchial mucous membrane, has long ago determined the fallacy of this argument. For myself, I think clinical experience is the convincing common-sense proof of the unimportance of the bronchial arteries as a source of hæmorrhage. Inflammation of the bronchial walls—for bronchitis is not merely an affection of the mucous membrane—is one of the very commonest of diseases. Yet how often has any one of us seen anything



approaching serious hæmorrhage in the course of this malady? Probably the most that we have perceived is no more than red streaking of the sputa, moderately stained sputa, or on one occasion, perhaps, a drachm or so of unmixed blood.

I fully agree, then, with the dictum of my old teacher, Dr. Reginald Thompson, who says: "All the evidence I can find shows—(1) The pulmonary circulation is the usual source of hæmoptysis, slight or serious; (2) the pulmonary circulation is the only source of profuse hæmoptysis, except in aneurism of the aortic circulation or the existence of a hæmorrhagic diathesis; (3) anatomically and pathologically the evidence is all against bronchial hæmoptysis."

The vascular anatomy of the lungs may be thus briefly described:

The pulmonary arteries do not anastomose, but the capillary system into which they break up does so very freely—a condition bearing importantly on thrombosis. The pulmonary veins have a sectional area smaller than that of the pulmonary arteries; they possess no valves and they freely anastomose. Their smaller area increases the blood tension within them: this, with the absence of valves and the free anastomosis, makes venous reflux very easy and powerful when the pulmonary artery is blocked.

The pulmonary capillaries have an aggregate sectional area less than that of the systemic capillaries; therefore the blood current must be the swifter in them. Moreover the calibre of each individual

capillary of the lungs is greater than that of its systemic co-relative. It is evident, then, that the rupture of a pulmonary capillary will lead to a more serious hæmorrhage than that of one belonging to the systemic circulation. Their network is one with extremely fine meshes : according to Kuss three-fourths of the surface of the alveoli being covered with them. The terminations of these capillaries are found to a great extent to be in the inter-lobular tissue, hence the pulmonary artery is a nutrient vessel as well as a secretory one.

The bronchial arteries arise from the aorta or the inter-costals, they do not anastomose with the branches of the pulmonary artery, but they do communicate with the capillaries of this vessel, for these can be readily injected from the bronchial arteries. Hence some of the blood from the bronchial arteries passes into the *pulmonary veins*.

Some of the blood coming from the bronchial *capillary* system also passes into the pulmonary veins. Thus in two ways the blood of the bronchial arteries enters the pulmonary circulation. This intimate connection would make us suspect that the bronchial system may be able to take on secretory as well as nutrient functions, and Virchow has described a case where it was enlarged consequent upon an impediment in the pulmonary supply. This likewise explains the great tendency there is to bronchial congestion in mitral disease, even when there is as yet no failure of the right ventricle.

There is a third system of vascular supply to the lungs. Branches from the œsophageal, mediastinal and pericardial arteries enter the hilum of the lung and spread out in the inter-lobular connective tissue. It has been found that if the pulmonary and bronchial arteries are ligatured the pulmonary and bronchial capillary system can be injected through these vessels.

Litten has shown us that the pulmonary circulation is very stable during the constantly varying vasomotor changes of the aortic system. It lives a life apart, undisturbed by the conflict of emotions, digestion, exertions and leucomaines which perplex and harass the existence of its burly brother.

Once more, Lichtheim tells us that even if as much as three-fourths of the lumen of the pulmonary artery be occluded, yet the same amount of blood will pass through the remaining one-fourth in any given time as before, owing to increase of pressure and its wonderful elasticity.

These two observations bear very strongly on the question of hæmoptysis and its treatment.

Such, then, is the vascular system, disease of which may produce hæmoptysis. What are these hæmoptoic diseases?

*Inflammation of any kind* is a most powerful pre disposing agent.

It has been shown experimentally that if a healthy vessel require a pressure of 70 mm. Hg. to produce extravasation, then the same one inflamed will give way under a pressure of 25—that is, with about one-third

the strain. Further, changes in the structure of the capillary walls must exist in the early stages of the inflammatory processes to allow diapedesis. It is probably only an excess of such changes which permits extrusion of the red cells, even to such an extent as to amount to actual hæmoptysis. Indeed, I think we can very fairly estimate the acuteness of an attack of pneumonia by a microscopic examination of the alveolar contents, and noting the proportion of red and white corpuscles found therein; for the greater the proportion of red cells, the acuter—*i.e.*, the more severe in its onset—the attack of pneumonia.

Yet inflammation of the lung parenchyma cannot alone produce hæmoptysis in any notable quantity, however acute it be. How rare is this in acute pneumonia—*e.g.*, to the extent even of a teaspoonful, and in amounts exceeding one ounce it is practically unknown. The vascular system appears to offer very much greater resistance to the ravages of inflammation than do the tissues in which it lies; but, unfortunately, not always to the same extent. Sometimes a comparatively feeble resistance to the surrounding inflammation is offered; the vascular walls become infiltrated with the inflammatory products and even the lumen itself of the affected vessel becomes blocked with them; solution of continuity occurs, and hæmoptysis-producing hæmorrhage results. Such weakness does not depend upon the nature of the inflammation so much as upon a predisposition previously acquired. (Dr. Donald Hood reports several cases of considerable hæmoptysis—half-

pint or more—occurring early, sometimes as the first symptom, in acute pneumonia (*Lancet*, 18 iii. 94). Isolated cases have also been reported by others. This occasional occurrence would strengthen the argument that it is not due to the disease, but to the predisposition. Still, I must admit that most of these cases are not followed up with sufficient persistence to definitely exclude the existence of tubercle and may, after all, be only exemplifications of the wisdom of the well-known axiom—Acute pneumonia with much hæmoptysis is probably tuberculous.)

This predisposition appears to be quite distinct from that to which we have given the name of hæmophilia, for this latter occurs chiefly in childhood, and is not confined to any one portion of the body; whilst the subjects of hæmoptysis are usually of adult age, and are not prone to loss of blood elsewhere. It has been spoken of by some, rather unhappily I think, as an hereditary or acquired fragility of the pulmonary vessels.

*Thrombosis producing an Infarct* is not an uncommon cause of hæmoptysis. The amount of blood lost is not usually large, and a fatal issue directly from the bleeding is extremely rare. This condition arises from various states of cardiac weakness, but, once established, the pathology is identical. The pulmonary arterioles being non-anastomosing terminal vessels, the portion of lung beyond the block is deprived of all blood from the pulmonary artery, and also, which is the more important event from the hæmoptoic point of



view, of the pressure which is derived from the pulmonary artery. The blocked area is now only supplied with blood from the bronchial, œsophageal, and other small vessels. These are sufficient to keep the area full, but have not enough pressure to maintain a proper flow against the reflux pressure from the veins, which (venous) pressure is maintained by the other unblocked branches of the pulmonary artery. Stagnation occurs ; the affected tissues, including the walls of the involved vessels, necrose, and hæmoptysis often results ; or the proximal portion of the artery containing the original thrombus may give way, and then a serious hæmorrhage, having behind it all the force of the pulmonary artery, may ensue.

It is evident, therefore, that the hæmoptysis does not occur at the moment of the occlusion, but only after an interval, during which vascular degeneration has taken place, allowing extravasation of blood. Clinically, too, we find this to be so, for if immediately after a spitting of blood in, say, a case of mitral stenosis, we examine the chest, it is very rarely indeed that we shall be unable to discover localised signs of pleurisy and of inflammation of the lung parenchyma—that is, signs of degeneration in the area of the infarct.

*Excessive Strain* of an otherwise healthy but weak heart may, though rarely, bring about a similar condition of things. Exhaustion of the left ventricle impedes the flow from the lungs into the left auricle ; slowing and stagnation of the blood in the lungs ensue, perhaps over a wide area, or, by inducing thrombosis, over two or

three smaller ones, and, in a day or two, hæmoptysis arises. It is noteworthy that in all the records of fatal hæmoptysis from severe strain which I have read, the spitting of blood never occurred immediately after the exertion.

*Injury without Puncture* may so contuse a portion of lung that inflammation and suppuration result, when, if a vessel be involved, hæmoptysis may occur. I have now in the Queen's Hospital a little girl who was knocked down by the shaft of a cart hitting her just below and outside the right nipple. On the following day rather profuse hæmoptysis occurred and continued for ten days; then—according to the history of Dr. Haynes, of Evesham, who sent me the case—she had an attack of pneumonia of the right lung, from which after three weeks she appeared to be convalescent. But a fortnight later, with a sudden fit of coughing, a large quantity of pus was expectorated, with the usual signs of abscess of the lung. Later, signs of pleural effusion with blood-stained serum in small amount arose, at which time she came under my care. She is now fast recovering, seems perfectly well in herself, and the chest exhibits the usual signs of contracting cavity with much thickened pleura.

The explanation is evident. The blow contused the lung parenchyma and so damaged the walls of one or more vessels that they gave way, hæmoptysis resulting. During the hæmoptysis pneumonia, arising from the contusion, crept on, and, by the time the hæmoptysis had ceased, had fully declared itself. This pneumonia

was of such severe type that a portion of the inflamed lung necrosed, became isolated, and broke down gradually into pus, which latter found its exit by a bronchus. Meanwhile, however, some inflammatory material was conveyed by the lymphatics or by direct extension to the pleura and set up pleurisy with effusion.

*Hæmoptysis from Metastasis*—e.g., vicarious menstruation—is very difficult for me to credit, unless there be some previous damage or hæmoptoic predisposition in the lung. That most wonderfully accurate physician, Walshe, thus expresses himself on this point: "With imperfect menstruation, in every case I have observed, except three, where the hæmoptysis has reached an ounce or more, there has been either evidence, or ground for the suspicion, of tubercle." Fagge is similarly sceptical. That such cases do, with extreme rarity, occur we may all admit; and, indeed, that patient of Pinel's of whom Sir Thomas Watson tells us, who after the first menstruation, which was normal, though suddenly suppressed by a fright, had hæmoptysis of the same amount every year, from 16 to 58—with the exception of one long interval—and with no apparent detriment to her health, obliges us almost to allow the possibility of such a condition.

The parasites causing hæmoptysis are three: *Filaria*, *Hydatid*, and *the Tubercle Bacillus*.

The first need not detain us here.

Of the second, I once had care for over two years of a typical case in whose sputum hooklets were from time to time discovered: this sputum was frequently stained,

and at intervals of a month or two pure blood was brought up for several days together, but never in amounts of dangerous character. The bleeding was no doubt due to destructive inflammation set up by the hooklets and also, previous to the rupture of the hydatid, by the interference with the circulation induced by the growing cyst causing stagnation and its accompanying inflammatory disturbance, such as I have previously detailed.

But it is in association with the tubercle bacillus that the great mass of hæmoptysis occurs. I say "in association" designedly, for that there is no indissoluble bond between the spitting of blood and the bacillus is evident in the fact that more than half the cases of tuberculous phthisis run their course without any hæmoptysis; in the fact that in acute pneumonic phthisis and in acute miliary tuberculosis of the lungs, two diseases where the bacilli lead the most virulent life and bring hasty death, hæmoptysis is most rare; in the fact again that in other primary lung affections, such as emphysema and fibroid disease, where bacilli play no *rôle*, yet hæmoptysis is at least as common as in those two previously mentioned, where they play their greatest *rôle* of all; and, finally, in the fact that hæmoptysis unites with the bond of a common symptom these primary diseases with those which are purely secondary to cardiac mischief, a mischief which is supposed by many to be positively inimical to bacillary life.

Yet hæmoptysis and tubercle are too closely

related to be looked on as mere concomitants, however much stress we may lay on the very great frequency of tuberculous phthisis as compared with all other diseases of the lungs.

Indeed, they both depend upon a common cause : the “*dispositio catarrhalis*” of the old writers, which I may best define as a tendency to a sluggish catarrh occurring in places where the circulation is feeble. The circulation is not sufficiently feeble to bring about degenerative inflammation and the resulting hæmoptysis in previously well-disposed tissue, as we have seen may result from that far greater enfeeblement of circulation which follows cardiac failure ; nor is the catarrhal tendency sufficiently active to start into being where the circulation is energetic, as occurs with the inselective catarrh of pneumonia. But where the catarrhal tendency and the feeble circulation both exist, there we have a constant focus of depressed activity, gifted with a dogged persistence which the strongest tissue must in time give way to, a veritable quicksand where it wallows helplessly, and in which at last, after passing through the stage of fatty degeneration, it dies suffocated. Such a slough of despond is the birthplace of hæmoptysis and the rich foster-mother of the bacillus.

All hæmoptysis in tuberculous phthisis arises from a perforating ulceration of a vessel's wall, or from the solution of its continuity after fatty degeneration and necrosis, or, by far the commonest of all, from the rupture of an aneurysmal dilatation of it.

Hæmoptysis is universally recognised as being very



rare in childhood : no doubt owing to the healthier condition of the tissues and the keener activity of the circulation. Tuberculous hæmoptysis is rare also because the cavernous stage of phthisis is rare, the lung usually suffering from discrete tubercle in conjunction with other organs. When resident at the Children's Hospital, Pendlebury, Manchester, I saw three cases of sudden copious fatal hæmoptysis in children aged 9, 4, and 1 years respectively. This last case was remarkable in that there was no evidence of tubercle outside the lungs except caseous peritoneal and bronchial glands. The right lung was emphysematous, with many masses of consolidation, but no cavities. The left upper lobe was a mass of breaking-down catarrhal pneumonia, with caseous masses and emphysematous patches. The left lower lobe chiefly consisted of a large cavity with thick irregular walls and with ragged projections, containing a few large exposed vessels, passing into it. The artery causing the hæmorrhage could not in this case be determined, though in the other two it was : but there was no doubt that a ruptured vessel, which was exposed or aneurysmal, had been the cause in all three. In the one-year-old child definite lung symptoms of dyspnœa and cough had existed for nineteen weeks : a very long time for so young a patient.

**Treatment.**—Granted my position as to the pathology of hæmoptysis the rationale of its treatment is simple, and the best means to carry out this rationale easy of acquirement. It will be best to consider these means under two heads : (1) when the hæmoptysis

arises from lung mischief secondary to cardiac failure ; and (2) when it is due to primary lung disease.

*The Hæmorrhage occurring in Cardiac Disease* is due to a degeneration of the endothelium, with consequent thrombus, in a branch of the pulmonary artery owing to feeble circulation, thus causing an infarct ; the hæmorrhage itself arising from necrosis of a vessel or vessels in the infarcted area from inflammation in the tissues surrounding it. But the tension of blood is very low, the effused blood quickly clots and lies against the opening, the blood within the vessel looks on this clot as a foreign body and forms a thrombus upon it, which soon occludes the bleeding point. The indication in cardiac hæmoptysis is therefore to prevent a repetition of the primary thrombus, and to effect this we have to strengthen the heart by horizontal rest and such tonics as strychnine and digitalis ; personally, I find subcutaneous injections, or rather intramuscular ones—for it is high time we did away with the term subcutaneous—of liquor strychninæ in the doses we are accustomed to give it by the stomach, 3 to 5 minims thrice daily, to be the most efficacious of all means for stimulating the weakened musculature of the heart.

Should the initial cardiac hæmoptysis be very severe we must at first have recourse to the treatment I am about to suggest for hæmoptysis due to primary lung disease, but even then we must be very chary in adopting measures which depress the heart.

*In Primary Lung Disease* we should look upon the bleeding point much as a surgeon would look upon a

bleeding vessel in a sloughing ulcer of the leg which he had to treat without the aid of any surgical skill. I take it he would be very ill-advised to employ ergot or any other vasomotor contractor, for the vessel in the neighbourhood of the bleeding point is diseased, its muscle fibres are degenerated, and therefore the drug will act less upon them than upon the fibres of all the other arteries of the body; the injured arteriole it will not contract, but it will raise the pressure of the blood within it by its effect on all the healthy arterioles; the result will therefore be an increase rather than a lessening of the bleeding, unless, indeed, you should so press your ergot that the general contraction resulting produced heart failure and in this way lessened the flow of blood at the bleeding point, though not to so great an extent as would occur elsewhere. I do not think this procedure will for a moment commend itself to us, it is a far more dangerous and a far less efficacious method of producing heart failure—that is, faintness—than Nature's own. She does it by bringing the tension of the blood to a minimum, and the blood ceases to gush from the extreme weakness of the propelling force; there is the rest of sheer exhaustion, and during it protoplasmic and chemical (not dynamic) forces create a plug for the hole in the relaxed and flaccid vessel. All the arterial system is in a state of half-empty flaccidity, so that the feeble flickerings of the heart have every encouragement to continue, for Nature has provided that it shall have the least possible work to do. And so life is kept going with the very lowest blood

pressure, thus allowing the plug the best possible chance of establishing itself. But in ergot-faintness the blood, so long as it moves at all, must move under considerable pressure, and hence plugging will be much less likely to take place. Moreover, the strong contraction of the arterioles gives the worn-out heart far less rest, and it has to re-establish the circulation in the least, instead of the most advantageous, circumstances.

As to ergot itself, I think I have given it a fair trial. My routine treatment when resident medical officer at the General Hospital was 30 grains of Bonjean's ergotine injected deeply into the muscles of the buttock. I never saw any injurious effect, local or general, result from such treatment; nor can I say that I was ever able to definitely associate any cessation in the hæmoptysis with its use. But even were it good to contract the arterioles of the lungs, how do we know that ergot will do this? As I stated before, the calibre of the pulmonary vessels is under very different governance to that of the systemic ones, and I am aware of no evidence tending to show that ergot can contract them.

Venesection is, I think, sometimes of value at the outset. If the pulse be strong, the individual full-blooded, or if venous congestion exist, then it is easy to see how a timely venesection may diminish the loss of blood. For half a pint of blood withdrawn quickly will lower tension, &c., as much as a pint removed slowly—that is, the quick withdrawal of blood by venesection may do at once what Nature is striving to

accomplish more slowly—namely, produce a faintness of the circulation.

Again, anything which keeps the blood in the systemic circulation, and so produces anæmia of the lungs, is beneficial. We know that the splanchnic area can contain all the blood of the body; any therapeusis, therefore, tending to fill this area must be good. Large doses of the nitrites, which relax the systemic arterioles, should thus prove of service, as well as the constant supply of small portions of food, so as to keep up a constant demand for blood in the alimentary tract.

To fulfil the same end ligatures may be applied to the thighs and upper arms, to prevent the blood reaching the right heart, a procedure well spoken of by Walshe. Leeches to the anus or a hot foot-bath may prove similarly useful. Till faintness comes on a posture only semi-supine is preferable; in this position the head is less well supplied with blood, and so less able to stimulate the heart's action than if the horizontal position were maintained.

Any means which may increase the coagulability of the blood is evidently of value. It is for this reason that the swallowing of gallic acid is recommended, though the method of its action is unknown to me. Nor do I think the evidence in its favour is considerable. That it does reach the blood in these cases is shown by the fact pointed out by Wood, that the blood spat up after its exhibition often has a greenish hue.

In January 1893, Professor A. E. Wright, of



Netley, made a communication to the *British Medical Journal* on the value of calcium chloride as an increaser of the coagulability of the blood. He showed, also, that in too large doses it delayed greatly the period of coagulation. In cases of chronic bleeding—for example, hæmophilia—doses of 5 grains taken thrice daily were most beneficial ; but to produce a sudden and complete effect, such as would be our desideratum in severe hæmoptysis, a large single dose of half a drachm would obtain the maximum effect which, in the professor's own case, reduced the coagulation time of his blood from 4 to  $1\frac{3}{4}$  minutes.

In the same journal, July 14, 1894, Professor Wright has made a further communication substantiating the effect of calcium chloride in cases of sudden bleeding, but showing that after internal administration for a few days—three to fifteen—its efficiency ceased, and the coagulability of the blood became even less than before. This I have found to be so in two cases of hæmaturia in which I used this drug. Hence, as Professor Wright points out, if we wish to increase, *e.g.*, the clotting of an aneurysm, we must give it for two or three days only, then omit it for a similar period, and then administer again, and so on. In the same paper Professor Wright shows the great power the inhalation of carbonic acid has in increasing the coagulability of the blood, and also that alcohol—*e.g.*, half a pint of champagne—diminishes this quality considerably.

Of all measures for the relief of hæmoptysis perhaps the induction of vomiting has the strongest clinical

value of any, but the rationale of its action is certainly obscure; and, though I have thought much about it, I am in no position to offer any elucidation of the problem. It matters little what drug be employed for the purpose, whether it be a local irritant, such as salt and water, or one acting on the centre, such as antimony. Personally, I prefer antimony, as it produces relaxation of the systemic arterioles as well.

Hydragogue purgatives are of extreme value, as they deplete the abdominal organs, and so enable them in turn to drain other parts of the body. Amongst them calomel possesses a double virtue, as mercury is one of the best, if not the best, drugs we possess for lowering arterial tension. Its great antiphlogistic power adds still more to the efficacy of mercury.

The great thirst which often accompanies prolonged bleeding is best assuaged by the sucking of small pieces of lemon, although Professor Wright has shown (*loc. cit.*) that citric acid lessens the coagulability of the blood. The exhibition of fluid drinks, except as nutriment, is to be deprecated, our aim being to keep the quantity of the blood as low as possible and its quality as concentrated.

A cool atmosphere is supposed to be advantageous. There is no doubt it is so in the case of epistaxis, and the nose is one portion of the respiratory tract. So it is hoped that it will be so to the vessels of the lung parenchyma, though we must remember—first, that, whatever the temperature of the outside air may be, that in the air-sacs is constant; and, secondly, cold air

is a stimulant to cardiac action, an action we wish to depress. Hence I believe a warm room to be preferable.

I must not forget one important aid with which Nature has provided us; she has given us a most capable ligature in the elasticity of the lungs. As blood is effused and more blood from the right heart takes its place within the vessel, the effused blood distends those alveoli into which it flows, and the elastic walls of these in turn increase the pressure which they had previously exerted, and so press the effused blood with increasing force against the ruptured wall of the vessel. This natural ligature it is evidently our duty to aid by all the means at our disposal.

I would shun any method of therapeusis which would accentuate the act of inspiration, such as the inhalation of astringent sprays.

The application of an icebag to the præcordium, inasmuch as it is a powerful cardiac depressant, is valuable, but, in so far as it chills the surface of the body, it is undesirable. As ice in some form or other is usually importuned for by the patient it is, perhaps, well at once to resort to this method of its exhibition, and the bag will be most wisely placed upon the præcordium. As to its local application over the bleeding point, even if we could accurately localise this, I doubt the advisability, owing to our want of knowledge of the effect of cold on the pulmonary vasomotor nerves and of the depth to which the cold of an icebag can penetrate. Experimentally, but not yet to my mind

clinically, it seems to have been shown that the cutaneous application of ice to the chest has produced local anæmia of the subjacent lung.

The last of eight admonitions prefixed to the rules of my school ran as follows : “ Above all things let the fear of God be before your eyes.” So would I conclude this paper with the exhortation : Above all things let the thought of morphia be ever in your minds.

As a chain’s strength is that of its weakest link so is the hæmoptoic force of a circulation that of its strongest pulse. One strong beat may thrust away the plug which many feeble predecessors have allowed to form. A vascular serenity is, therefore, an absolute essential in the treatment of hæmoptysis ; so, then, let our first care be to produce in our patients an opiate calm that they may, in the words of Matthew Arnold, be—

“ Undistracted by the sounds around them,  
Unaffrighted by the sights they see.”

## XI. THE ANTISEPTIC TREATMENT OF TUBERCULAR PHTHISIS

THE complete antiseptic treatment of phthisis has four main ends to accomplish—viz. :

(1) The prevention of the entrance of the bacilli of tubercle into our bodies or into the bodies of domestic animals.

(2) The destruction of the bacilli which exist in the waste products of the disintegration of tissue induced by their action.

(3) The destruction of the bacilli which exist in the still living tissues.

(4) To nullify the septicæmia which results from the ptomaines and various pyogenic germs, which arise as secondary products of bacillary life.

The first of these belongs to the domain of prophylaxis and lies chiefly in the province of the medical officer of health. To achieve it we must scrupulously disinfect the dwellings previously inhabited by sufferers from tubercle, and especially all articles of personal use. Close physical intimacy of life between phthisics and non-phthisics must be strenuously striven against, and



most of all must we, as physicians, with persistent pertinacity conduct a crusade against the marriage of bacilli-laden organisms, never tiring in our efforts till women have included such marriages in the code of their immoralities. The hidden evils of sputum must be no longer kept shrouded, but fully exposed to the view of the laic mind till all men and women are aware of the wickedness of ubiquitous spitting. It is by thus awakening the moral sense of the laity that, in my opinion, lies the great hope of tubercular prophylaxis : the disease is much too insidious to be dealt with successfully by legal restrictions alone ; and we must never forget that the duty of pioneering in this awakening is ours, as disciples of medicine.

To attain the second end, complete sterilisation of the sputum is necessary, and also the disintegrated tissues must be made innocuous before they become sputum, whilst they lie in the cavities of the lung caused by their disintegration, or in the lumina of the bronchi. We thus not only destroy the evil power of the sputum, but also render harmless the air expired, and prevent the auto-infection of other regions of the body—*e.g.*, the larynx—by means of the air passages. The sterilisation of the sputum can be easily and completely accomplished, and there is therefore no excuse for its omission, nor do I believe that if the laity were only fully aware of the immense importance of its performance, there would be any difficulty in getting them to thoroughly carry it out, for they show us an excellent example in the willing energy they display in obeying the regulations concerning

isolation and disinfection in specific fevers. But as yet the medical profession itself is a very poor leader, as the unhygienic state of the spittoons in many of our general hospitals abundantly testifies. There seems great hope that the sterilisation of the excretion before it leaves the body is by no means impossible, either through inhalation of suitable remedies or by injections into the cavities of disease, or, at least, the upper air passages may be kept in a state refractory to the bacillus by similar topical methods, and so auto-infection of these be prevented. This last, considering the rarity of primary laryngeal tuberculosis and its rebelliousness when once started, is a precaution too little considered by us.

The third end, that of destroying the bacilli in the still living tissues, is the real aim we have to strive for in the antiseptic treatment of patients suffering from tubercular phthisis. There are several ways of attaining this: we may (*a*) endeavour to make an antiseptic solution of the blood and lymph; (*b*) increase the cardiac strength so that there may be a more vigorous circulation through the bacillary districts; (*c*) enhance the respiratory interchange of gases, thus keeping the body as thoroughly oxygenated as possible; (*d*) increase the nutrition of the cells of the lung parenchyma; (*e*) increase the positive chemiotactic\* power of the bacilli and also the capacity of the phagocytes for

\* Chemiotaxis is the term given to the property micro-organisms (or rather the soluble products of these) possess of attracting (positive chemiotaxis) or repelling (negative chemiotaxis) the wandering cells (phagocytes).

devouring bacilli, since Hankin has shown that it is one thing to bring the phagocyte and bacillus together and quite another to excite the phagocyte's voracity.

To nullify the septicæmia of phthisis (our fourth end) we must destroy the pyogenic germs which cause it. Jakowski (*Centralbl. für Bakt.*, 9, xii. 93), has examined the blood of nine patients suffering from phthisical hectic: in five he found staph. pyogenes aureus; in two, staph. pyogenes albus; in three, streptococcus pyogenes. These were sometimes alone and sometimes in combination; in two patients no cocci were found. In one case, where no cocci were found at first, when there was no hectic and the disease was limited to one apex, at a later examination, contemporaneous with a further destruction of lung tissue, they were readily obtained. Phthisical hectic, it is evident, must therefore be treated on the same principles as we treat pyæmia arising from other causes.

In this paper I shall only attempt to discuss the capacity of some drugs to make the body fluids antiseptic as regards the bacillus of tubercle. This is an action altogether apart from phagocytosis. Phagocytosis is the destruction of a germ by a living cell and is a variety of protective inflammation; whereas the action of antiseptic drugs is a purely chemical one and consists in poisoning the bacillus but not necessarily dealing in any way with its dead body. So far as I can learn, no drug has yet been shown to possess the power of destroying the vitality of all the bacilli existing in the

lungs of a human being the subject of tubercular phthisis, though there is good evidence that during the exhibition of some, the bacilli are placed temporarily *hors de combat*. If this is so, it is evident the disease will be arrested so long as the drug is administered, and there seems ground for hope that after a prolonged administration all these paralysed bacilli will have been eliminated from the body.

There are many drugs—*e.g.*, creasote and eucalyptus—which will purify foul expectoration, will prevent the occurrence of decomposition within it, in other words, will kill the bacilli of various kinds—including the *B. tuberculosis*—which may be contained in it. But this is a very much easier thing than destroying the germs in the lung parenchyma itself; just as it is far easier to render sterile a typhoid stool than to cut short the multiplication of the germs in the walls of the ileum. Expectoration, whether it be in a lung cavity, in the bronchi, or in the spittoon, is merely the excrement of diseased lung, an excrement which has become so greatly in excess of the normal that it can no longer be got rid of by normal means—*viz.*, by exhalation and re-absorption.

It is then by its power of destroying the bacilli in the still living and still vascular tissues that the value of antisepsis has to be judged. I am one of those who believe it has great capacities in this direction, though at the same time admitting that no drug can compare with the anti-bacillary force of the phagocytic action of the organism itself.

For the purpose we have in view drugs can be



administered by inhalation, intra-pulmonary injection, intra-muscular injection, or *per os vel rectum*.

Inhalation is the feeblest of all methods for administering antiseptic remedies to the lung parenchyma. If these be inhaled in the form of pulverised liquids it is doubtful if they ever reach beyond the medium-sized bronchi. In the gaseous form, it is true, they may be taken up by the blood, as is oxygen, and by means of the lymphatics may attack the bacilli in the parenchyma. Fraentzel, in 1883, and Hillier, in 1885, tried inhalations of carbolic acid, creasote, bromine, sulphuretted hydrogen, iodoform and others, but in no instance found any diminution in the number of bacilli, nor any change in the morbid condition of the lungs. As we shall see later on, other investigators have been apparently more successful, though probably most of the benefit derived from inhalations is due to their action upon the products of tubercular disintegration rather than upon the *cause* of this morbid process.

The *locus standi* of intra-pulmonary injection I fail to discover, nor can I admit the reasonableness of its employment unless we believe that a much stronger solution of a drug can be injected into a focus of diseased lung tissue than can with safety be passed into the general circulation by means of the muscular lymphatics. But clinical experience refutes this belief: indeed, the same solution often produces more untoward symptoms when injected into the tissue of the diseased lung than when injected into muscles. And this is indeed what would be expected, for the circulation in



the diseased focus is enfeebled and to a great extent cut off, whereas the drug injected into healthy muscle is speedily and greatly diluted by the rapidity of the fluid currents which it enters : moreover, muscle is notably less vulnerable than lung parenchyma. Intra-pulmonary injection seems to me to stand on the same footing as excision : the scope of both must ever be extremely limited : limited to those patients where we have very greatly preponderating evidence that the tuberculisation is absolutely confined to one well-defined focus, and the physician who has ever felt confident that he has, in any case, elucidated such evidence has attained a more fortunate experience in pulmonary tuberculosis than has, as yet, fallen to my lot. To inject an antiseptic into a tubercular *cavity*, no matter how circumscribed it be nor how sure we feel of its singleness, is, to my mind, but playing with a great therapeutic measure and falsifying its results ; for post-mortem examinations indubitably show that wherever a cavity exhibiting signs of activity has existed there also have been found, in the tissue around it, many or few, grey and yellow tubercles, to most of which the solution would not penetrate, as Dr. C. T. Williams' experiments on dead subjects tend to show. If, on the other hand, the cavity be stationary and quiescent or contracting we should surely do best by acting on the old adage, "Let sleeping dogs lie." Results confirm these conclusions, for though post mortems have shown that in some cases the cavities have been benefited, yet the very fact of these post mortems negatives the value of the treatment.

Intra-muscular injection is no doubt the best way to reach the blood and lymph, and there is no lack of suitable solutions to administer in this way, but it is not a pleasant mode of administration and we English have not as yet taken kindly to it for chronic disease. Moreover, the strong association of the hypodermic syringe with morphia makes us hesitate before we place such an instrument in a patient's hands ; and, unless we do so, the method is beyond the reach of the majority of phthisical patients.

There is left to us the alimentary tract. The mouth being the natural entrance for food is evidently the most congenial gate through which to take any solid or liquid into our bodies, but its use has the serious objection of sometimes causing gastric upset. The rectum has not this drawback, and most drugs can be well absorbed by it, and if given in suppositories not much objection is made by the patient ; hence it is a valuable alternative to the mouth in those cases where gastric disturbance arises.

The group of *Phenols* has perhaps been used in the treatment of phthisis more than any other antiseptic, and I will proceed to discuss this first. Pharmacy has now reached a step beyond the crude drugs, carbolic acid, creasote, and guaiacol, which from their caustic poisonous nature and unpleasant taste were very difficult of administration, and has passed to compounds of these, which, whilst harmless and tasteless, are almost equally powerful as antiseptics. Such are salol, betol, benzosol, paracresalol, guaiacol carbonate, creasote car-

bonate, and the group of bismuth phenols, of which naphthol-, cresol-, and tribromo-phenol-bismuth are the most important.

The members of the phenol group destroy all germs in solutions outside the body, as well as those existing in the alimentary tract; but during absorption they change into unknown non-antiseptic compounds, for Hoelscher (*Berl. Klin. Wochens.*, No. 3, 1892) has shown that the blood cannot be sterilised however large the dose exhibited. These phenols then cannot be strictly termed antiseptics as regards living germs in the tissues, but there seems good reason to believe that after absorption they combine with the albuminous toxins which result from germ life, forming non-toxic compounds, which are eliminated by the urine as ethereal sulphates. They do not kill the bacilli, but they obviate the evils resulting from the existence of these,\* and thus prevent injury to the tissues, the bacilli remaining harmlessly in the body, and in due course becoming excreted, we would hope, as effete material; but no clinical proof of the extermination of the bacilli by the use of phenols has been produced, though there is ample evidence of the power they possess in keeping in check the evil symptoms of tubercular septicæmia.

*Creasote* itself is an old remedy for tuberculosis. It was thus used by Reichenbach so long ago as 1830. Bouchard and Gimbert revived its use in 1877, publish-

\* For, as I have before stated, it is the toxic albumins which result from bacillary life which attract the phagocytes—that is, produce *pneumonic* phthisis.

ing an exhaustive analysis of 93 cases so treated. They found :—(1) a lessening of cough and expectoration in one to two weeks ; (2) in three weeks a diminution of the fever with an increase in the appetite and strength ; (3) at the same time, frequently, the night sweats vanished, and the disease appeared arrested ; (4) a few weeks later, improvement in the physical signs indicative of fibrosis appeared, this sometimes occurring with almost incredible rapidity.

Sommerbrodt aroused a fresh German interest in the matter in 1887 by claiming it as a specific, basing his statement on an experience of nine years and 5000 patients. He usually exhibited it in a capsule containing  $\frac{3}{4}$  of a minim with  $\mathfrak{m}$  3 of tolu balsam four to six times a day.

Bouchard and Gimbert gave as much as 11 minims in a single dose. But Warner (*New York Med. Journ.*, 11, xi. 93) has gone far beyond this amount ; in one case the patient, a lady, taking for a long period a daily dose of 78 minims. Warner gave it after food in milk, sweetened water or wine, or with gentian or cinchona. He found that the general condition greatly and rapidly improved, the cough lessened, the sputum became more watery, and the fever and sweating often vanished. Sickness and diarrhœa were the chief symptoms of an overdose with premonitions of headache, vertigo, and dimness of sight.

In my own experience, creasote acts as a gastric irritant in any quantity beyond a daily dose of  $\mathfrak{m}$  6, and I have never traced any change in the pulmonary and

constitutional condition of my patients to its use in such amount. But I have never thoroughly tried the drug as, when pushed, it proved so disagreeable.

*Guaiacol* forms 60 to 90 per cent. of beech creasote (Martindale) and possesses the latter's antiseptic qualities. It is far less unpleasant to taste and smell than creasote, and I have found patients take it with comparative ease dissolved in cod-liver oil in doses of 2, 3, and 5 minims three times daily. Only occasionally have such doses disagreed, and I have noted distinct improvement in cough and expectoration as an almost uniform result, yet other changes have not been sufficiently constant to enable me to ascribe them with any certainty to the action of this drug.\*

But guaiacol is already out of fashion and I do not suppose it will ever again have a vogue for phthisis. So butterfly-like is the life of a drug! A descendant, *Guaiacol Carbonate*, has usurped its position. No doubt its usurpation is permanent, for it has none of the evil qualities of its ancestor, whilst possessing, apparently, all its power for good.

It passes through the stomach unchanged, but in the intestine it slowly resolves into guaiacol and carbonic acid, the guaiacol in its nascent state being absorbed as quickly as it is set free. The small amount of carbonic acid set free acts merely as an intestinal sedative, just as

\* Since the above was written I have had a young lady who for three months has taken a daily dose of  $\mathfrak{M}$  75 of guaiacol, and who still continues to do so. For a few days she took  $\mathfrak{M}$  100, but this produced flatulent malaise.



it does when given with hydrogen sulphide enemata. Guaiacol is excreted in the urine, though not as guaiacol, but as a body which reacts like phenol with bromide water (Poggi, *Rif. Med.*, Aug. 10, 1892). This body is found in the urine half an hour after the ingestion of the carbonate. Guaiacol carbonate is an odourless, tasteless powder containing 91 per cent. of pure guaiacol. As much as  $2\frac{1}{2}$  drachms have been given daily with no untoward result. I have usually begun with 5 grains thrice daily and have not gone beyond a dose of 10 grains. There is no doubt of its good action on the catarrhal element in phthisis, but I have not yet observed any removal of deposit—it has merely *dried* the lungs. However, my experience has been both scanty and brief, only extending over twelve months. Its expense almost debars it from use in the out-patient department, as *a priori* it would take large doses to fully try it by making the body fluids thoroughly antiseptic.

Reckoning the bacillicide action of guaiacol as equal to that of carbolic acid, and 5 per cent. to be a sufficiently powerful solution of this latter: further, estimating the body fluids of an ordinary patient to weigh 20 pounds, then we should require to maintain constantly in the body one pound of guaiacol, and as it is so rapidly excreted\* after ingestion, the maintenance

\* Linossier and Lannois found it in the urine fifteen minutes after its application to the skin: the maximum excretion occurred from one-and-a-half to four hours after the application, and only traces could be detected in the urine twenty-four hours after. 55·5 per

of this amount would probably require 4 ozs. daily to be administered ; though that in the lymph would be much less quickly eliminated than that existing in the actual blood stream itself.

Can such an amount be taken? A ten-weeks-old puppy is said to have taken 5 drachms daily, and Hoelscher gave as much as 100 grains daily to patients with no obvious ill-effects. Indeed, a dose of  $2\frac{1}{2}$  drachms can apparently be daily ingested with impunity. Guaiacol carbonate has evidently not yet been pushed to its limit, but whether that limit will be so far beyond the doses at present administered as 4 ounces it is impossible to say.

Could such a dose be given, how long would the treatment have to continue? The spores of bacillus anthracis are destroyed by a twenty-four hours' exposure to a 5 per cent. solution of carbolic acid, according to experiments made by Koch.\* In early tuberculosis, where the tubercles are small and therefore no bacilli or spores far removed from the active circulation of blood and lymph, two or three days of such "disinfection" should prove sufficient. After making full allowance, especially in later cases where the caseous masses are large, for defective circulation, still a week of such thorough treatment should suffice to destroy any germs existing in the body.

cent of the amount applied was recovered from the urine. (*Sem. Méd.*, 7, ii. 1894, quoted by *Brit. Med. Journ. Supp.*, 17, ii. 1894.)

\* Brunton : "Pharmacology and Therapeutics," p. 74. Brunton also states that "the action of carbolic acid on other fully developed microzymes, or on the spores, is almost the same as on the anthrax bacilli" (p. 75).

The eradication of tubercular phthisis by a single week's treatment makes one think of Utopia rather than of this work-a-day world ; yet in medicine truth has ever proved stranger than fiction, and *a priori* there is no inherent impossibility in the matter whatever. I have never been able to fall in line with those thinkers who maintain that any antiseptic strong enough to destroy germs within the body will also destroy the body cells. If the body cells can resist the injurious influence of an antiseptic no more than germs, then surely the whole fabric of antiseptic surgery as well as of antiseptic medicine falls to the ground ; for on this reasoning, if the antiseptic do not destroy the cells it will not destroy the germs, and no one carries antisepsis in surgery to the point of cellular necrosis. But yet who can deny the specific action of iodoform in the local treatment of tubercular abscess ?

Moreover, if this theory hold good, then any smaller dose of an antiseptic must be futile, for whatever damage it may inflict upon the germ it will inflict equally upon the phagocyte ; *both* combatants will equally suffer, and hence no good accrue to either. I can perceive no logical *via media*. It cannot be argued that any hindrance to the inroad or multiplication of the germ must correspondingly strengthen his opponent, the phagocyte. This is not so, for the very cause of the mobilisation of the phagocytic army is the invasion of the germ, and the greater the host of germs the greater also the army of mobilised phagocytes. The result of the warfare is a sad desolation of the lung

parenchyma—the battlefield of the contending forces ; but whether this desolation be chiefly due to the ravages of the germinic, or to those of the phagocytic army, it is impossible to say.

Another perhaps more practical question is the advisability of slowly or rapidly increasing the dosage ; by slow increments more can, in the end, be usually tolerated, the system becoming habituated and more refractory to the action of the drug. But do not the germs get similarly refractory ? And we must remember that the generations of germs in any given time will be more numerous than the generations of cells. Natural selection would thus tell in favour of the germs ; they would become immune to the drug sooner than would the cells. If there be any cogency in this argument, it will be wise to rise to the maximum dose as speedily as possible.

*Hydrofluoric Acid* is an antiseptic possessing great capabilities, but it is difficult to speak of it with any certainty, for I have discovered very little literature concerning it during the last six years, and most of what follows is drawn from a Report made by M. Hérard, its President, to the French Academy of Medicine in November 1887.

As an antiseptic Hayem considers it on an equality with iodide of mercury. At the Manchester meeting of the British Association in 1887, Wm. Thompson stated that both it and sodium fluosilicate were most potent antiseptics. Dujardin-Beaumetz and Chevy placed samples of water, urine, and various broths in glass

vessels. To some of these samples they added hydrofluoric acid, making them solutions of 1 in 500, 1 in 1000, and 1 in 2000 respectively. After nine days none of the liquids so treated had undergone the slightest change, whereas all those liquids to which no acid had been added began to putrify on the second day.

Again, to putrid solutions (six days old) of meat and urine they added 1 in 1000 of this acid, and in two days they had all become perfectly sweet and clear.

H. Martin (Report to French Academy, September 1887) inoculated six Pasteur flasks of veal bouillon with tubercle. Two of them were set aside as controls. The other four had 1 in 5000, 1 in 10,000, 1 in 15,000, and 1 in 20,000 hydrofluoric acid added to them. All the flasks were kept in a temperature of  $38^{\circ}$  to  $39^{\circ}$  C. In fifteen days both the control flasks contained a flocculent proliferation of bacilli, whilst in the other four the bacilli were all destroyed : a result which Martin states is superior to any he has obtained by any other antiseptic. But he noticed two curious circumstances : (1) if the solution were allowed to become neutral or alkaline the germs grew even faster than they did in the control flasks ; and (2) if four days after the addition of the acid the bouillon was inoculated, the bacilli thus admitted were able to grow, though but slowly ; this no doubt resulting from the action of the acid on the glass with the formation of an inert silicate.

Martin further inoculated guinea-pigs with tuberculous material. Those animals who were kept as controls all became tuberculous, dying in twenty to forty



days. Two other pigs were inoculated with a portion of the material to which 1 in 5000 of the acid had been added : one of these died in an hour of acute peritonitis and the other only lived a few hours longer. One in 10,000 of the acid was added to the injection given to two others : these both died in six weeks of subacute non-tuberculous peritonitis. To four others, injections were given containing 1 in 50,000, 1 in 70,000, 1 in 80,000, and 1 in 100,000 respectively : none of these had peritonitis, but they all died of tubercle.

Hérard inoculated guinea-pigs and rabbits with tubercle, some of these he kept as control subjects, and when killed these had *areæ* of acute congestion around the tubercles which had arisen, as indeed is usually the case. The others he placed in a box through which he passed a stream of air containing hydrofluoric acid vapour. On examining the bodies of these he found no *areæ* of acute congestion around the tubercles ; moreover, many of these latter had undergone fibrous and cretaceous change. One of the rabbits which were inoculated had young ones, all of whom died. After their birth the mother was placed in the box and submitted to the hydrofluoric treatment under which she greatly improved. She then again had young ones, and all these lived and were healthy.

Experiment therefore seems to show that this acid possesses great bacillicide powers. Moreover, the active principle appears to be the fluorine. For Martin made use of a 1 in 100 solution of ammoniac fluoride. He inoculated three rabbits with tubercle and kept one

as a control. The other two had m 6 of ammonium fluoride injected subcutaneously every day. The control animal died of tubercle in fifteen days ; one of the others died in twenty-four and the other in twenty-seven from the effects of the ammonium fluoride, for in neither was any tubercle found.

In this connection it is interesting to note a statement of MM. Arthus and A. Huber in the *Archives Générales de Médecine*, 1893, vol i. p. 98. They say that a 1 in 100 solution of fluoride of sodium stops completely all fermentations due to germs—*e.g.*, the lactic, the ammoniacal fermentation of urine, the alcoholic, &c. ; but that it has no action on chemical fermentations—*e.g.*, those of the digestive ferments. They have used this drug to diagnose between these two varieties of fermentation, and assert that the glycogenic function of the liver must be chemical in nature since fluoride of sodium has no effect upon it.

Bergeron's is, I think, the most practicable method of administering the gas. This method, which is described in the *Journal de Médecine de Paris*, February 1888, is briefly as follows :—The patient is placed in a wooden chest of two cubic metres capacity. Into this chest the diluted gas is admitted through the roof at a uniform rate of two litres a minute. At the end of every fifteen minutes the patient steps out of the chest, which is then thoroughly ventilated for five minutes. Each *séance* may occupy two, three, or even more such quarters of an hour. The diluted gas Bergeron obtains by passing

fresh air through a leaden Woulff's bottle containing either the strong acid or, preferably, a mixture of sulphuric acid and fluor spar. Both tubes of the bottle are short so that neither dips into the solution ; the air therefore does not pass through this, but simply takes up the gas contained in the upper part of the bottle. Commercial hydrofluoric acid, if of good quality, for this contains a quantity varying from 27 to 54 per cent. of the pure acid, will do sufficiently well. A strong acid must be used to ensure a definite amount being taken up by the current of air, for Bergeron found that fresh air took up four milligrammes of pure acid per litre of air both when it was passed very slowly over the commercial acid and also when it was passed at double this rate ; but if a dilute acid were used the amount taken up varied considerably with a variation in the current-speed, and also in other ways.

Garcin's plan of inhalation is the pleasanter, for he passes the gas from his apparatus into the air of any ordinary room in which the patient may be, striving to maintain the proportion of 20 to 30 litres of gas-charged air for every cubic metre of capacity the room contains. He orders the patient to stay in this atmosphere for one hour daily. But this does not seem so effective a method as Bergeron's, for the hydrofluoric acid being a heavy vapour would not diffuse so equally through the atmosphere as if admitted through the ceiling into a small chamber. Yet Garcin obtained excellent results : of 100 cases 10 died, 14 remained in *statu quo*, 41 improved, and 35 were "cured ;" these

cured cases having so remained without relapse for periods varying from fifteen to thirty months subsequent to the omission of the treatment. But both the cured and improved cases belonged, nearly all of them, to the first or second stage of the disease.

Hérard in his report states that he found nothing disagreeable in the atmosphere thus produced by Garcin, that the inhalations were incontestably beneficial, that they were free from inconvenience, easy to use, and could be combined with all other kinds of treatment.

It is this last sentence which puts those who are sceptical amongst us at once on the alert and prompts the question, How much of the improvement noted was due to the combination of other kinds of treatment and, one may add, how much to unaided Nature? The attitude of mind which leads us to ask these questions is a most proper one if to it be added the finer attitude which bids us prove all things. But without the desire for proof the doubt becomes but a lazy scoff utterly unworthy the scientific mind of medicine.

Here at any rate we have the President of the French Academy of Medicine making these assertions as regards the beneficial action of hydrofluoric acid upon patients who have been examined and watched by himself:

- (1) In a wonderful way, even perhaps after the first inhalation, the appetite returns and remains good.
- (2) Vomiting, if present, ceases with equal celerity.
- (3) Diarrhœa, though far more rarely and much less rapidly, is still sometimes improved.

(4) Night sweats, as a rule, diminish at once and soon cease.

(5) Pyrexia is soon modified, but takes long to go altogether.

(6) Dyspepsia is markedly relieved after eight or ten *séances*.

(7) Sleep and weight both greatly improve.

(8) The cough becomes less stubborn, less constant, and less spasmodic ; that occurring during the night being especially relieved.

(9) In laryngeal cases the pain of speaking is lessened, but the voice is only slightly improved.

(10) The expectoration lessens, becoming white, frothy, and mucoid.

(11) The bacilli vanish. Schmidt examined 17 of Garcin's patients, and in 16, after repeated observations, failed to find any. Twelve months after the cessation of the treatment he had failed to find any for six months in these 16. At Hérard's request Charrin checked Schmidt's results and corroborated them in all points. Seiler only examined one of his patients for bacilli. In this case, after a month's treatment, these had notably diminished and he failed to detect any elastic fibres.

(12) Finally the local changes in the lungs, though naturally of much tardier development than the amelioration of symptoms, distinctly show improvement and fibrosis.

The history of the introduction of hydrofluoric acid into medicine is an instructive one. Ever since 1820



the good effect the occupation of etching had upon workpeople with delicate chests had been recognised in the glass factories of Paris, and the glass cutters, who were peculiarly liable to phthisis, frequently asked to be allowed to take up etching instead of cutting. In 1862, Didierjean, a chemist in the large factory of Baccarat, made this fact known to a relative of his, the enthusiastic scientist, Dr. Bastien. Bastien prevailed upon Charcot and Bouchard to give the drug a trial: they did so for a couple of months, but obtained no success from its use and dropped it after this too hasty trial with a negative expression of opinion. However Bastien himself took up its use with avidity and made it a regular part of his armamentarium. Some years later he recommended its use in a case of croup which he saw in consultation with Bergeron. Bergeron used it and was so satisfied with its action that from 1877 to 1884 it was his sole remedy in such cases. In this latter year he made a Report to the French Academy on the subject. Its use in phthisis was first brought to the notice of the Academy by Seiler in July 1885, who again still more forcibly urged its use in an address at Nancy in 1886. In 1885 Chevy, a pupil of Dujardin-Beaumetz, wrote an able report on its properties and its favourable action on a few phthisical patients. Then in September 1887, Garcin recorded the results of its use in 100 cases under his own care, expressing his belief that it stood in the very front rank of phthisical remedies.

*Oil of Eucalyptus* has long been a favourite remedy

with me in phthisis. I was first drawn to it as a valuable drug from observing the excellent results it gave when used as a spray instead of carbolic acid in the dressing of empyemata, and especially from noting its sedative and healing properties when used as an emulsion in the dressing of chronic abscess cavities.

When physician to the Jaffray Hospital I used it persistently in cases of phthisis in doses of 2 to 5 minims dropped on sugar and taken three and four times a day. The results seemed to me superior to those obtained by treating similar cases under the same circumstances with cod-liver oil, or oil and malt, or with the somewhat similar drugs—terebene, pumiline, and pine oil. Especially was its action good in soothing cough by easing expectoration and in removing any accompanying bronchitis. Generally, the local condition also improved, though the patients were not hopeful ones, being usually in an advanced stage and recovering from some acute attack either of hæmoptysis or pneumonia, for which they had been admitted to the General Hospital. But later knowledge has shown me that such doses were not large enough to obtain all the possible good of which this drug is capable.

In February 1884, Professor Ball, of the Laennec Hospital at Geneva, on the suggestion of Dr. Roussel, began to treat his phthisical patients with subcutaneous injections of eucalyptol. He continued to do so for three years, and then made a report to the French Academy, in which he stated that in his cases diarrhœa, cough, expectoration, night sweats, and fever all im-

proved, and that these improvements were followed by an increase in the general health and appetite. In one of his patients, after the treatment no bacilli could be found in the sputum, though they were zealously sought for during three months.

Regarding this question of bacilli, Hainaut (*Journal de Médecine de Paris*, June 1888) states "that eucalyptol alone of all the methods we have tried prevents the multiplication of the bacilli." Roussel goes to the extreme of optimism, for he writes (*Union médicale*, 1887): "As to bacilli, after two or three months' treatment, the severest examination fails to find any trace of them. This exciting cause has completely vanished." On the other hand, Dujardin-Beaumetz says (*Acad. de Méd.*, March 1887): "Never have I seen the bacilli vanish under the influence of these injections. Eucalyptol modifies and lessens expectoration; it is a balsamic drug possessing the advantage of acting without causing gastric disturbance, but it is no specific against tuberculosis." Bouveret and Péchadre (*Lyon médical*, Feb. 1887), Biot (*idem*, May 1887), Laplane (*Marseille médical*, 1887) and Guiffart (*Union médicale*, June 1887) are, on the whole, in agreement with this declaration of Dujardin-Beaumetz. As I have indicated above, my own observations at the Jaffray Hospital, made contemporaneously, are quite in accord with those of these observers.

But Bouveret and Péchadre, whilst acknowledging the good action of eucalyptol on the catarrhal symptoms, assert that in febrile cases it is not only of no

avail, but is positively harmful. Here, it seems to me, their *a priori* theory has biassed their clinical observation, for in my hands it has benefited both pyrexial and apyrexial catarrh, though it is manifest that catarrh, or any other morbid manifestation, occurring in the septicæmic body of a hectic phthisic must needs be less amenable to treatment than the same symptom arising where this septicæmia is absent, or, if present, exists in so slight a form that it is unable to modify the general body temperature. However, having determined that the drug had no action on the bacilli or the poisons emanating from these, they also concluded that it could not lessen any evil symptom arising in a body which was constitutionally suffering from such infection.

Hainaut, on the contrary, asserts that the blood is so rapidly purified that pyrexia and night sweats vanish about the eighth injection. The two statements are not irreconcilable. If the hectic be due to an increasing deposit of tubercles eucalyptol is not likely to diminish it, but if it be due rather to the removal of caseous *débris*, a hectic in fact of ordinary suppuration, then eucalyptol will probably lessen it just as it lessens the hectic of an empyema.

But after all, is not this discussion as to the presence or absence of bacilli in the sputum somewhat vain and wide of the mark? I do not suppose any one of us has sufficient hardihood to declare a case of pulmonary tuberculosis cured so long as there be *any* expectoration, whether this do or do not contain bacilli. And if there be no sputum, how can we examine for bacilli at all?

It may be asserted that expectoration may persist from the lowered pulmonary tone, inferior tissue, and impaired circulation, which are but the results of the ravages of a past tuberculosis. This may indeed be so, though as yet we have no sufficient clinical and pathological material to speak definitely concerning it. But even granting it, yet for all practical purposes we shall be assuredly well advised to treat such cases as only "improved" ones, for we must ever remember that tuberculosis confers no immunity as do the specific fevers; on the contrary, one attack renders the body far more susceptible if the local defences in the lungs have been permanently injured thereby, for the bacillary foe is ever present to act on the offensive.

And let me here impress upon you most strongly the fact that absence of expectoration is no evidence of absence of bacilli. Time after time have I been thus disappointed; for months all expectoration has been absent, both the general and local conditions have steadily improved, and I have hoped that a permanent cure was in progress: then, at a next visit, one mouthful of sputum has been brought me, the patient may or may not complain of a slight cold, and I may or may not be able to detect a small patch of moisture; but whether or no, very seldom do I fail to find the fatal germ in the expectoration given me. But such disappointments should not induce in us despondency, rather a fresh outburst of energy; for is not the progress of syphilis, in the curability of which every one believes, liable to the same unexpected and tantalising relapses!



There are several species of eucalyptus. *Eucalyptus globulus* has the best reputation. The peppermint gum tree, *E. amygdalina*, is a stunted form which grows on poor sandy hills and dies on marshy soils. *E. drumosa*, the mallee scrub, is a low bush which grows on the dry plains of Australia. *E. oleosa*, or mountain ash, has also been employed in medicine. Dr. Benjafield, of Tasmania, says no aboriginal Tasmanian has ever been known to use any one of these three for healing purposes, whereas, even when Tasman came to the island, he found the leaves of *E. globulus* extensively chewed by the natives. Nor have these ever been planted in new districts (*e.g.*, California) as antimalarials. *E. globulus* oil should be heavy (sp. gr. 0.9 about) and green : it should also be quite free from phellandrin and other terpenes. The other species are lighter, more spirituous, and more closely resemble turpentine. But of *E. globulus* itself there appear to be several varieties as well as impure specimens of these varieties : so that at present one is more likely to get an impure or imperfect sample than one possessing the full medicinal value of the pure oil. The pure oil has about 41 per cent. of eucalyptol, which is usually considered to be the active principle, and it should not contain any constituents irritating to the mucous membrane of the bronchi. This latter quality places eucalyptus on a higher therapeutic level than pine oil, terebene, turpentine, &c., which irritate the bronchi by spasmodically contracting their vessels, as well as above guaiacol and the various phenols which cause

bronchial hyperæmia by inducing vasomotor dilatation.

Eucalyptus is generally admitted to be a powerful germicide : its action on full grown bacilli is from three to five times as strong as that of carbolic acid ; but its action on spores is only a weak one, probably considerably less than that of carbolic acid. The germicidal power does not reside in the eucalyptol alone, for Omelchenko has shown that while the vapour of eucalyptol takes 134 hours to kill the bacilli of anthrax, 72 hours exposure to the vapour of eucal. glob. oil is sufficient to destroy them. Indeed, eucalyptol bears something the same relationship to eucalyptus oil that morrhuol does to oleum morrhuæ and, antiseptis apart, we must at least lose most of the balsamic virtues of the oil by the use of eucalyptol. Benjafield quotes a letter to the *Tasmanian News*, wherein a gardener states that " if fruit trees be mulched in winter with eucalyptus leaves they will be entirely free from blight and fungi " during the ensuing summer. He also tells us that he injected a little of the oil into a rose tree covered with aphides, and that these all disappeared in a few days.

France, the land of hypodermic medication, gives a daily injection of some 4 minims dissolved in vaseline (12 minims). Hainaut states that this medium both weakens the antiseptic action and produces local irritation. So given, it is perceived in the breath at the commencement of the treatment in fifteen to twenty minutes and the odour persists for a few hours only, but after a few days, in three or four minutes the patient

smells it and continues to do so throughout the period during which the injections are administered. Inhalations have not this great drawback to such a degree, but in one of my patients the persistence of the odour after an hour's inhalation seriously impaired the appetite and almost necessitated omission of the drug. Except in disease affecting the larynx it is best to give the oil by the mouth in capsules of  $\text{m} 5$  to  $\text{m} 10$  each, the dose being gradually raised till  $\text{m} 30$  are taken thrice a day. Exhibited in such a manner its prolonged use produces no ill effects, and can be relied on to greatly relieve the catarrhal element, at any rate, of tubercular phthisis.

*Sulphur* is a classical remedy for phthisis pulmonum, for Galen recommended his patients to live in huts near the crater of Vesuvius so that they might inhale the sulphurous acid fumes which were given off.

Sulphur mineral waters have long been of good repute in the treatment of phthisis. They are divided into the soda sulphurs, as those of Cauterets, Bagnères de Luchon, Amélie-les-Bains—lime sulphurs, as at Enghien and Schinznach—both lime and soda, as those of Eaux Bonnes and Harrogate—and the famous springs of Allevard, which are somewhat nearer the lime than the soda springs in constitution. Besides sulphuretted hydrogen, which is common to all, the soda springs contain nitrogen, and the lime carbonic acid. The action of all these waters is chiefly to stimulate the organism as a whole, and especially the pulmonary mucous membrane. They are not to be used in acute phthisis which they might only intensify,

but in the chronic forms they produce a healthy stimulation which at first increases and afterwards dries up the expectoration. It is wise to begin drinking the water cautiously—not more than a quarter of a tumbler thrice daily—and afterwards to carefully increase the dose according to the effect brought about. Baths should also be employed when possible, these acting much as a mild universally applied mustard plaister might be supposed to.

The introduction of *Sulphurous Acid* into modern medicine as a remedy for phthisis bears a striking resemblance to that of hydrofluoric acid, for it was occasioned by the observation of the good effects that were produced upon phthisical employés in factories where fumes of this gas were constantly evolved.

The *Lancet* reported in 1883 a statement of Kircher's that he had never heard of any phthisis among the employés of the factory of which he had been director for forty years, and in which large quantities of sulphurous acid were produced. The managers of several other similar factories supported this statement.

In 1887, Dr. Auriol, an enthusiastic French physician, found several phthisical patients at work in an atmosphere made most irritating by the fumes of sulphurous acid. He wished to have them removed to other parts of the factory, but they begged to be allowed to remain, expressing firm belief in the efficacy of the fumes for the alleviation of their disease. Thereupon Auriol at once submitted seventy phthisics

to the following treatment :—He placed the patient in a corner of a large closed room and lighted a little sulphur at the opposite corner. In the atmosphere thus produced he kept the patient till a moistened litmus paper began to redden. This was done twice daily. Thirty early cases were arrested and their sputum would no longer infect guinea-pigs. Twenty obtained no benefit and twenty would not persevere.

Ley has shown that many animals can well support an atmosphere containing 1 : 2000 sulphurous acid ; but that 1 : 200 was toxic to most. Similarly a healthy man could with difficulty support a 1 : 6000 proportion ; and he thinks the therapeutic dose should be from 1 : 10000 to 1 : 8000

To obtain this dosage Dujardin-Beaumetz proceeds as follows :—Let the room be thoroughly ventilated and filled with fresh air. Light a quantity of sulphur equal to 70 grains for every cubic yard of capacity. Do this in the evening, and closely seal up the room. The next day let the patient live in this room from 7 till 11 and from 2 to 5. The reason for lighting the sulphur in the evening is to allow time for the gas to thoroughly diffuse and for the vapours to be condensed. This takes ten to twelve hours. After the first three days, when the walls and furniture have become thoroughly soaked with the gas, 30 grains of sulphur will suffice.

This sulphurous acid treatment has been tried to a great extent in Paris, and apparently with much success and with no ill effects. Cough and expectoration are



always lessened and sleep improved. The most striking instance I have read is a case recorded by Dr. Sollaud in 1887.

A sergeant of marines at Cherbourg, age 26, after a severe chill had, in June 1885, a copious hæmoptysis. In October, Sollaud found well-marked signs of phthisis. In February 1886 he returned from a long sick leave considerably worse, with the signs and symptoms of extending cavitation. Under careful tonic treatment his general health much improved, but the local condition remained unchanged. Numerous bacilli were found in the sputum.

Wearying of hospital life, by much importunity he obtained leave to return to duty. At this time it happened that the barracks required thorough disinfection. Fortunately for him, to this sergeant fell the duty of opening the doors, windows, &c., of the sealed rooms in which the sulphur used for disinfection had been burnt. At first he bitterly complained of the irritation of the sulphurous acid, which produced great pulmonary oppression with much increase of expectoration and cough. But Sollaud persuaded him to persevere in his duty. Soon he found he could breathe without inconvenience in the densest fumes, and his condition began greatly to improve—"beyond all expectation," says Sollaud. Six weeks from the commencement of the disinfection the following note was made :—Cough and expectoration almost gone. Diarrhœa and hectic have vanished. There is no dyspnœa ; the appetite is good, and he is daily gaining strength. The number of bacilli is much diminished. Slight signs of consolidation at either apex.

Four months later (*i.e.*, September 1886) there were absolutely no signs of pulmonary disease—no bacilli had been found for a month, and he expressed himself as being thoroughly well ; and so continued up to the time of Sollaud's report to the Académie de Médecine in March 1887.

*Sulphuretted Hydrogen* Dr. Pilatte has found to be both the most powerful agent in preventing the development of the bacillus and also the most active destroyer of the fully developed germ. Hanging for one minute a piece of blotting-paper soaked in a solution of tubercle in a current of this gas rendered the paper harmless.

Dr. Niepce, a physician at the sulphurous spa of Allevard, placed some tuberculous sputum in one of the drinking rooms for twenty minutes. He then found it had no effect upon guinea-pigs, though some of the same sputum, not so treated, produced tuberculosis in some other guinea-pigs in thirty days.

He also injected four mice with tuberculous matter. Two of these he kept for six weeks in the drinking hall, then killed them and found no evidence of tubercle in them; the other two he kept in the open air, and they soon died, having their viscera filled with tubercles.

Lastly, he injected four rabbits with tuberculous material; two he kept in the fresh air and they died tuberculous. To the other two he gave inhalations of pure sulphuretted hydrogen for thirty minutes thrice daily for three weeks; and, when, after a while, he killed them, he found them quite healthy.

This same indefatigable Dr. Niepce gave a patient with moderately early phthisis an inhalation of 3 per cent. of this gas for fifteen minutes four times daily. He had previously proved that his sputum was actively tuberculous by inoculating a guinea-pig with it, and also by finding numerous bacilli in it. During the treatment

the bacilli steadily lessened, and after twenty-seven days he tried in vain to inoculate another guinea-pig.

Renzi stated in 1885 that he found inhalations of sulphuretted hydrogen as valuable as those of sulphurous acid, but others have not been so fortunate, and the gas has never been much employed in this fashion.

In 1857 Claude Bernard proved that sulphuretted hydrogen injected into an animal's rectum was eliminated by the lung with great rapidity before it had time to reach the systemic arterial circulation, and that the gas thus given produced no disturbance in the animal's health.

To Bergeon of Lyons occurred the idea of utilising this fact in the treatment of tuberculosis. But he added carbonic acid to the sulphuretted hydrogen, as he found by experiment that the two gases could be injected together with impunity, and he hoped that the sedative action of carbonic acid would neutralise the unpleasant peristalsis and flatulence which sulphuretted hydrogen alone not seldom set up. Bergeon was very particular in insisting upon using a natural water as his solution of sulphuretted hydrogen ; and Fraentzel declares that with an artificially prepared gas he has failed to obtain any benefit.

The Bergeon method is simple—carbonic acid is obtained from sulphuric acid and sodium bicarbonate—this gas then passes into a Woulff's bottle containing a solution of sulphuretted hydrogen ; from this bottle the mixed gases are injected into the rectum by a modified Higgensen's syringe.

The regulation dose, according to Bergeon, is four

litres of the mixed gases, and fifteen to twenty minutes should be taken to inject them.

In most cases, in a few days, Bergeon found a marked lessening of cough, sputum, and dyspnœa. Sleep improved. A sense of well-being arose; and appetite, strength and weight increased. But in no case did the bacilli vanish. Many other experimenters have in the main agreed, but the treatment has never found favour in England, chiefly owing to the malodour of the gas, but partly to an Englishman's dislike to frequent disturbance of his anal sphincter.

Dr. C. T. Williams tried it on five cases at the Brompton Hospital, but was unable to get the patients to submit to the procedure with any persistency, and so abandoned his trial.

Mr. Bremner of Leicester carried out the treatment on three private patients in 1887 and 1888—Bergeon's cases were published in July 1886. One of these I saw with him in consultation. There was chronic infiltrating tubercle at both apices. The sputum was scanty, but in it Dr. Crooke found considerable numbers of bacilli. The patient, a spare, pallid, married man of 30, caught cold in May 1887, and thereafter suffered from cough, streaked sputa, night sweats and emaciation.

The sulphuretted hydrogen treatment was commenced on October 15, 1887, and continued till December 24. Three pints of gas were administered every night, and thirty minutes were occupied in the administration. He improved and the temperature became normal. Towards the end of January 1888 he had a severe

hæmoptysis (30 ozs.), and it was after this that I first saw him. He expressed great faith in the beneficial result of the treatment, and after a minute examination I failed to detect any mischief at the left apex ; but the upper part of the right lung showed signs of small scattered foci of consolidation, though in a dry state : the sputum still contained bacilli. This patient soon after went to Melbourne, taking a position in some bank there, and I have just heard (Sept. 1894) from Mr. Bremner that he is and has always remained healthy.

Of the two other of Mr. Bremner's cases one was a girl of 17, whose mother had died of phthisis. For six weeks four to five pints were injected night and morning. At the commencement she had an evening temperature of  $103^{\circ}$ , with much emaciation and abundant expectoration. Extensive and advanced disease in the left lung, with a left pleural effusion and early disease at the right apex. The temperature became normal, and she gained 6 lbs. in weight ; while the cough and expectoration decreased. The improvement continued, and eight months later she had gained 9 lbs. more, was in a good general condition, only complaining of a cough, while both sides of the chest had much flattened over contracted lung. But three years later she relapsed and died.

The third case was a schoolboy, 17 years old and six feet in height, who developed phthisis after typhoid. This patient also lost his pyrexia, gained weight, expectoration ceased and cough became slight. He went to Cambridge : is now a tutor in good health (1894).



*Tuberculin* I cannot pass by without some notice, however scant this be. There is something intensely dramatic in the suddenness of its fall from popular favour. But this is because it ought never to have achieved the position it did, and only gained it through the *éclat* of its introduction by so illustrious a parent as Koch. It is now fast passing into the limbo of drugs which have been tried and found wanting. Yet the oblivion which is shrouding it cannot be owing to its proved inefficacy, for the results asserted to have been obtained by it are at least as good as those obtained by any other remedy. Its neglect seems rather to be due to two positive forces—first, the dangers of its exhibition in even careful hands; and second, the discovery of various modifications of the original poison. Even so, I do not think we are warranted in dismissing it from practical medicine so long as such reports as those of Von Rück, Schiess Bey and Kartulis, and Thorner are forthcoming. Thorner (*Deut. Med. Wochen.*, September 14, 1893), after a personal experience of two years, believes in it; even in advanced cases he has obtained improvement in cough, expectoration, diarrhœa, fever, and night sweats. He has seen no ill effects due to its proper administration; but this observation is rather self-condemning, for he further states that anorexia, pallor, or pyrexia occurring later than the fourth week of treatment should lead to immediate disuse of the remedy. His method was to begin with one-twentieth mg. every forty-eight hours for ten doses. Then steadily increase, reaching one mg. at the end of

four weeks. Then, each increasing by one-fifth mg., give two doses a week, till two mgs. are reached. Finally, after a rest of three weeks, give one injection of five mgs. You will notice that these are far smaller than the doses in vogue during the Tuberculin-Berlin *furore* of 1890-91.

Schiess Bey and Kartulis (*Zeit. f. Hygiene*, xv. pt. 2) give the results of its action in their hands on forty-eight patients in Egypt, the climate of which they think peculiarly suited to this method of treatment. Compared with other cases living under similar conditions, the advantage lies with tuberculin. Its action is harmless ; commencing phthisis it cures ; and even advanced cases, if chronic, may slowly recover.

But no one has reported on tuberculin anything like as favourably as Karl von Rück. In the *Therapeutic Gazette* of June 15, 1891, he related the histories of twenty-five cases so treated. And again in the same periodical, two years later (June 15, 1893) he continued up to date the histories of these same patients. Thirteen were advanced, with considerable constitutional disturbance, though he considered none hopeless. Of these, three were greatly and three moderately improved, while seven had died. In seven others moderate local breaking down had occurred, but the constitutional state was good ; six of these he looked on as cured, and the seventh as greatly improved. The remaining five of his twenty-five cases had one or both upper lobes affected, but not beyond the first stage ; these were all cured.

He had previously reported, in 1890, on 515 cases

treated under similar conditions to the above, with the exception of tuberculin. Eighty-one of these had little constitutional disturbance, twenty of whom recovered and sixteen remained improved. Of the remaining 434 who were more advanced, thirty-nine recovered, and forty-eight remained improved. Putting these in percentage form, we get :

	With Tuberculin.			Without Tuberculin.		
	Cured.	Improved.		Cured.	Improved.	
Disease local only . .	92	...	8	...	24	... 21
Constitution affected .	0	...	46	...	9	... 11

Von Rück, like Thorner, begins with one-twentieth mg., which in his experience has never given a reaction. He then increases by one-tenth mg. each injection till one mg. is reached ; then by one-fifth mg. to two mg. ; by one-half mg. to five mg. ; by two-and-a-half mg. to twenty mg. ; finally, by five mg. to one decigramme if needful. The injection must never be repeated in less than twenty-four hours, and in no case till all reaction has vanished. If the reaction be great, it is best to return to the previous lower dose. When all signs of phthisical activity cease, then omit the injections and if there be no recurrence of these be content to simply watch the case. If signs again arise, the treatment must be again gone over from the beginning, starting with the initial dose of one-twentieth mg. After an experience of six to seven thousand injections on one hundred different patients, Von Rück states that he has never noticed any discomfort arise from them.

Ehrlich explained Koch's view of the action of

tuberculin at the International Congress of Hygiene held in London, August 1891. He stated that at every spot where active bacilli existed there was a certain amount of toxin produced by these, which set up a corresponding amount of irritation of the tissues, or, in other words, attracted a certain number of leucocytes; that the use of tuberculin increased the force of the leucocytic army of attack simply by being so much injected toxin added to the toxin which was already there as the result of the activity of the bacilli themselves. By the united efforts of the two toxins there was a greater chance that the leucocytes would be able to set up a more thorough inflammatory encapsulation of the bacilli than if only stirred to action by the slowly evolved toxin of the germs. This, then, is the problem to be solved: your injection must be sufficient to produce complete encapsulation, but it must not be sufficient to attract suddenly such a multitude of leucocytes as will break down the tissues they traverse, and so give greater freedom, instead of encapsulation, to the germs which they purpose to destroy. There seems little doubt that with the larger doses originally employed destruction of tissue was not seldom a consequence.\* Thus it is not claimed for tuberculin that it has any direct influence upon the bacilli, but that it

\* This destruction must be carefully distinguished from the clearing out of a localised deposit, leaving a cavity which speedily contracts—an event which is usually one of very happy augury. But in this latter case encapsulation has taken place prior to the clearing out, which is merely the removal of practically extra-vascular *débris*.



adds to their attractiveness in the eyes of the avenging leucocyte.

Ehrlich maintains further that the local hyperæmia produced by tuberculin sets up transudation currents into the practically extra-vascular tissue of the tubercle itself; hence bacillicide remedies, if administered during the period of reaction, had a greater chance of actually reaching the bacilli. Langenbusch used picric acid and mercuric chloride in this manner, and asserted that of ninety-nine unfavourable cases forty were improved and thirty-three cured.

*Tuberculocidin* is a modification of tuberculin introduced by Klebs in November 1891. It forms  $2\frac{1}{2}$  per cent. of tuberculin. For it he claims these advantages over tuberculin:—(1) It does not produce the profound cardiac depression. (2) The red corpuscles do not become adhesive, nor do the leucocytes get irritated; hence there is no local reaction. (3) When given to guinea-pigs in doses which would be equal to 3 drachms given to a man weighing 130 lbs., it does not cause more than  $5^{\circ}$  C. rise in temperature. (4) At the same time it is no indifferent substance, for 2 grammes may cause weariness and emaciation if daily administered. (5) It produces partial immunity, for after taking it guinea-pigs take twice as long as usual to develop tuberculosis. (6) It attacks the bacilli directly; often, even after a few injections, these become granular and degenerate and, if the injections be omitted, the germs regain their pristine vigour. (7) In animals, after its use, the highly developed tubercle



is absent ; with the microscope minute ones may be detected, but in these are no bacilli ; along with this happy result an excessive development of smooth muscle occurs in the bronchi and vessels.

*Tuberculin and Tuberculocidin.*—Carl Spengler, of Davos, though unable, experimentally, to prove the direct bacillicide action of tuberculocidin, agrees with Klebs in the main as to the clinical benefit derived from its use ; but he finds it lacking in activity. For instance, he never found any decided curative effect upon the ulcers of pharyngeal tuberculosis. A local irritation—*i.e.*, a phagocytic attraction—is needed to remove the tissue *débris* and so allow the tuberculocidin to act. Spengler therefore added tuberculin to the tuberculocidin to effect this purpose. This combination enabled him to give much larger doses of the tuberculocidin ingredient of tuberculin than if he had only given tuberculin alone. He found that he obtained reactions with much smaller doses of tuberculin than if he only used this drug alone—*e.g.*, with an injection of one-thirtieth mg. tuberculin plus one-thirtieth grm. tuberculocidin. Further, that he had no occasion to go beyond one mg. of tuberculin plus one-third grm. of tuberculocidin, and so avoided all danger arising from large doses of tuberculin. This treatment of Spengler's is evidently analogous to that of Langenbusch, which I have before spoken of—Spengler using tuberculocidin as a bacillicide in the same way as Langenbusch used picric acid and mercuric chloride.\*

\* Hunter's B modification of tuberculin appears to have much

Of *Iodoform* I have had considerable experience during the last eight years, and on the whole I consider it the most satisfactory of all the antiseptic drugs which have been used in tuberculosis. But I am now speaking purely as a clinician and with somewhat bated breath, for I am not unmindful that there are many who deny that it has any power in pulmonary tuberculosis and, indeed, some who even say it fights for, instead of against, the bacillus. Thus, Heyn and Rovsing, of Copenhagen (*Fortschritte der Med.*, 1887, ii. p. 39), say that never have they found the mixing a relatively large amount of iodoform with a tubercular inoculation has prevented the development of general tuberculosis in rabbits, and that in two instances they believed it quickened the onset of tuberculosis at the point of inoculation. Again, Filleau and Léon Petit, in their interesting Bulletins on Tuberculosis (No. 2, May 1887), state that it has no influence on the vitality of the tubercle bacillus, not even when the inoculated spot is the eye, where one would have thought the access of light would have aided the setting free of iodine and intensified its action. But more than this, these French *savants* add that the drug appears to have an irritating action on the tissues, which renders them peculiarly prone to the inroad of the bacillus of tubercle!

the same properties as tuberculocidin, and can be used in the same doses. Trudeau (*Med. News*, Phila., September 3 and 10, 1893) filtered the bacilli from a liquid culture of them. Injecting the bacilli he found produced no symptoms but those of depression and general chronic septicæmia; but injection of the liquid filtrate had much the same effect as a similar injection of tuberculin.

But Gosselin, as we shall see directly, in this same year 1887 came to a very different conclusion—to wit, that though iodoform did not kill the bacillus, yet it prevented the anatomical development of tubercles, it appearing to render the tissues sterile to the foe. And it is with Gosselin that the mass of clinical evidence goes, both surgical and medical; and with him, too, I throw in my lot. The researches of chemists point in the same direction. M. Rummo, in a valuable paper in the *Archives de Physiologie* for 1883, p. 144, found iodine in the urine one hour after the entrance of iodoform into the stomach, but the excretion went on so slowly that traces of iodine could still be detected in the urine three days later. The iodine seems to escape by all the secretions, partly as an iodide, partly as an iodate, and partly as a new organic compound of iodine. Iodine itself is eliminated in the same way. It would seem, then, that the iodoform first splits up into its components, and that in the act of excretion the iodine is again recombined. If, therefore, we recognise iodine to be a powerful antiseptic, it appears difficult to deny the same rôle to iodoform. However this may be, it has certainly kept its hold on the profession since it was first introduced as a remedy by Dr. Glover in 1837—fifteen years after its discovery by Serullas.

As long ago as 1852 Righini wrote a very complete monograph on the drug, praising its use as an inhalation in early phthisis, and expressing his belief that when taken internally it arrested the disease. At the Amsterdam Congress in 1879 Semmola reported his

experience. As a portion is excreted unchanged by the lungs, he concluded that by giving the drug internally he would obtain both the local and constitutional effect; he accordingly gave it in pills, but did not exceed a daily dose of 8 grains. Yet, and even with smaller doses, he found great lessening of cough, expectoration, fever and putridity, with a corresponding improvement of the local and general condition. Rummo obtained still better results by employing doses as large as 15 grains dissolved in 360 grains of essence of turpentine as an inhalation; he believed that the turpentine helped the local action of the iodoform.

Dreschfeld and Shingleton Smith are our chief English authorities. The latter, in 1884, presented a report of his experience with it to the Copenhagen Congress. He was "in the habit of increasing the dose from time to time from 1 grain up to 5 or even 6 grains five times daily," but his average maximum, judging from his table of cases, appears to have been 3 grains thrice daily. In only six out of forty-six cases did improvement fail to occur; increase in weight and fall in temperature being the two most striking results.

M. Stchegoleff finds that beef-peptone-gelatine bouillon, which is an excellent culture medium for Koch's tubercle bacillus, loses that property when it has incorporated with it 5 per cent. of iodoform, the tubercle bacilli sown in it dying in forty-eight hours. Also, when an emulsion of virulent culture of the bacilli plus 10 per cent. iodoform was employed to inoculate

guinea-pigs, the animals survived longer than those which had received non-iodoformed inoculations (*Archives de Médecine Experimentale*, November 1894).

It was the excellent results obtained by its use in the Manchester Children's Hospital, where I was House Surgeon in 1883, that first drew my attention to its value. Strangely enough, though I have never used it since with anything like the freedom I did then, yet I never knew a child there suffer from symptoms of poisoning.

Soon after my appointment as Assistant Physician to the General Hospital, I began to try the effect of iodoform upon the phthisis among my out-patients. Through the courtesy of the hospital authorities I have been able to examine my case books for 1886, 1887, and 1888, but, unfortunately, of the many instances where iodoform was used as the main treatment, I can only collect 46 cases where the notes were kept with sufficient persistency to make them worthy of record. Of these 12 were much improved, 15 improved, 11 remained the same, and 8 grew worse—that is, 59 per cent. improved and 17 per cent. grew worse. It will be seen that no deaths are recorded; this is owing to the exigencies of a multitudinous out-patient *clientèle*, which made it almost impossible to follow up those patients who became too ill to attend the hospital.

Of the 12 who much improved 6 had reached the third stage, but only 2 of these had both lungs attacked, and only 1 amongst the earlier cases was so affected. Of the 15 who improved, 11 had reached the third stage and 7 had both lungs involved. Five of



those who grew worse had both lungs attacked, and 3 of these had reached the third stage. Of the 11 who remained stationary, 7 had reached the third stage and 5 had both lungs diseased. You will see from these statistics that the advanced cases did as well as the early ones. This I think is due mainly to the favourable nature of the advanced, these being mostly cases of localised cavitation or those in which considerable fibrosis had occurred. The unfavourable advanced cases felt themselves doubtless too ill to stand the exhaustion of out-patient attendance.

My usual prescription was a one-grain pill to be taken six times daily. It never exceeded this amount and was occasionally less, the average being 5 grains daily. In three cases only were any symptoms of poisoning detected, and these were of merely a mild gastric nature. I also tried the drug in conjunction with oil and tonics, and compared both classes of cases with those in which oils and tonics alone were used. The conclusion I arrived at after a three years' trial was a definite though by no means an extremely sanguine one. I believed that iodoform given by itself gave me better results than any other drug or combination of drugs that I had tried or seen tried. It soothed the nervous system of erethic subjects ; it very greatly lessened cough and expectoration ; it powerfully increased nutrition, the patients often becoming quite plump under its continued use ; finally, there was as great if not greater improvement in the physical signs than I had seen accomplished by any other mode of treatment, except that of climate and hygiene.

I think this improvement in nutrition and appetite is very striking testimony in favour of iodoform having some specific power over tubercular disease. For when given to healthy persons there is no doubt of its generally depressing effect, especially upon the nervous and digestive systems, producing gastric catarrh, inappetence and a condition of mental misery approaching melancholia. Whereas in tubercular subjects it frequently induces a feeling of well-being with a rapid increase of weight, comparatively large doses being taken with impunity. One of my cases has taken as much as 50 grains a day with no ill effect of any kind. Indeed, tubercular patients have seemed to me to feed upon iodoform much as syphilitic ones do upon mercury.

I have since used iodoform for the cure of phthisis in some private patients and in some in-patients at the Queen's Hospital, and my later experience fully bears out my previous impressions. Before analysing the results obtained, it will be as well perhaps to first tell at some length the story of a case which is remarkable in several ways.

C. H., schoolboy, 18, I saw with Mr. Leech who examined him one month previous to my visit on two occasions and found no sign of pulmonary mischief, in fact there had been no definite lung lesion detected till toward the end of April. On May 8, 1891, when I first saw him, the heart was greatly exposed, pulsation being marked in second, third, and fourth spaces. There were all the signs of widespread and rapid excavation in the upper part of the left lung, and throughout existed the large crepitation of tissue destruction. The right apex also showed signs of consolidation. Bacilli were numerous and the lad was tall,

thin and debilitated. His expectoration was profuse and mucopurulent, and he had severe constant pyrexia with night sweats.

Under such circumstances I could but share in Mr. Leech's prognosis of a speedily fatal issue, though I held out some hope on account of the well-known fickleness and uncertainty of the disease, especially as the family history was good. He was given Fellows' syrup and 2 grains of iodoform thrice daily.

On May 14th, his heart was much stronger, his dyspnœa less, his temperature lower, and his general condition improved. The left lung was drier and the base clearer. There were signs of extension on the right with the formation of a small cavity. He now takes 8 grains of iodoform daily and is gradually to increase the dose by a grain every other day.

On June 6th, he had no expectoration and hardly any cough. His n.s. had gone and his temperature was normal. The lungs had improved and the lad was heavier. His daily dose of iodoform is 20 grains and it is to be increased to 30.

August 18th. Has gained 8 lbs. since last note. Stomach reaches up to fourth space on the left. The heart is exposed up to the second cartilage. The lung is almost dry, and the cavities are much smaller. The right apex shows merely deficient P.N. and slight broncho-vesicular B.S. The right lung extends one inch to the left of the sternum. There was evidence, then, here of rapid contracting fibrosis of the left lung with corresponding hypertrophy of the right.

December 3rd. He has been two or three months in the Isle of Man. He has continued well, and his father wishes to know if he may join him in his business. He has persisted in taking daily 30 grains of iodoform. His weight has increased 16 lbs., an increase of 24 lbs. since his illness seven months ago.

June 6, 1892. Has continued well. No cough nor expectoration this year. Is still taking iodoform, an average daily dose of 27 grains. Still plenty healthy-looking bacilli in a small quantity of sputum he spat up one morning.

December 15, 1892. Is distinctly worse, though his lungs

remain dry and are more fibrosed. Plenty of bacilli in the morning expectoration which is only occasional. He has lost much weight during the six months, and has done so steadily, being now 18 lbs. lighter than he was in May.

February 21, 1893. Very feeble, with severe hectic fever, an acute attack having supervened, with evidently a fresh outburst of tubercle at the right base.

March 28th. Has continued very ill and wasted. On the 24th he had a large hæmorrhage, but still seems better than in February.

June 22nd. He recovered from the fresh attack, but never regained his strength. He is very fragile, weighing only seven stones—two stones less than he did twelve months ago.

October 1893. He dies, having persistently taken a daily dose of over 20 grains of iodoform, and for long periods 30 grains, during the whole two-and-a-half years of his illness, with no ill effect, so far as I could determine, of any kind whatever.

I have ventured to detail this case to you at some length because—

(1) It is a very good justification of the dictum that we should never give up hope in cases of acute phthisis. This is a dictum of whose truth I feel more and more convinced the wider my experience becomes. For six months of the thirty during which I watched this lad I should have been justified in calling his a case of arrested phthisis.

(2) It bears out in part the result of an experiment of Gosselin's in 1887. He gave some rabbits and guinea-pigs a daily injection of three drops of an ethereal 10 per cent. solution of iodoform. After some time he inoculated these animals with tubercle, and found that the evolution of the disease was notably delayed. In another series of cases he inoculated the animals first,



and then injected the iodoform daily into some of them, keeping the others as control subjects. These controls all died of tubercle between the thirtieth and forty-eighth days after inoculation. The others he killed on the ninety-fifth day, having given ninety-five injections, and found in them no trace of tubercles, not even in those in which he did not begin the injections for ten or twelve days after the inoculation, unless indeed the lesions were already far advanced by this time. But—and this is the point to which I wish to draw your attention—he never found that the bacilli were destroyed, for in several cases where he kept the animals alive after he had ceased injecting the iodoform, tuberculosis always sooner or later appeared and went on to its fatal termination. It would appear therefore that he merely made the tissues of these animals sterile so far as the bacillus is concerned. The bacilli were starved, but not slain. And we are well aware of the remarkable pertinacity with which the bacillus of tubercle clings to life. Or it may be that iodoform kills the bacilli, but is unable to destroy the spores, though these cannot develop in tissues saturated with the drug. So with my patient C. H. During the period of arrest—at least six months—bacilli were always found in any occasional sputum that was expectorated and examined. Yet during this same period rapid strides were made towards health and strength, both locally in the lungs and constitutionally—strides which would have been still much greater had not the heart been sadly upset by the dislocation induced by the rapidly contracting left



lung. The bacilli appeared to be quite harmless, and would probably always have remained so had not the destruction of lung tissue been too great to admit of due oxygenation of the blood sufficient for the wants of a growing lad. In all probability they continued to multiply in some region of diseased lung which was nearly cut off from vascular supply, and therefore from the iodoform conveyed in that supply.

(3) It emphasises the need to continue the remedy for a long period after *all* symptoms have vanished (*cf. syphilis*).

(4) The case is interesting as conclusively showing that a daily dose of 20 to 30 grains of iodoform can be continuously taken for two-and-a-half years with no detectable ill effect.\*

(5) It exemplifies the good results which iodoform is usually said to bring about, and which I have previously mentioned.

Ten other cases which I have abstracted from my case book I treated for periods averaging five-and-a-half months each with iodoform as the chief remedy. The initial dose was 6 grains daily, this being quickly raised to an average maximum of 15 daily. All these had but slight pyrexia, and were in a state of health sufficiently good to enable them to consult me in my rooms. The following are brief notes of their histories.

\* I must not omit to mention that drachm doses of Fellows' syrup of the hypophosphites were taken thrice daily for several months, and that cod-liver oil in varying doses he took during the greater part of his illness. These no doubt helped his nutrition, but I am convinced the iodoform was by far the most powerful remedy, and my patient was more firmly of this opinion than I.

Case.	Daily Dose and its Duration.	Condition at		Later History.
		Commencement.	End.	
1.	6 grs. 2 months.	III. Stage. Both lungs.	No change.	The same 6 months after.
2.	6 to 20 grs. 6 months.	I. Stage. Both lungs chronic.	Local signs less; much stronger; gain of 8 lbs.	Considers himself cured.
3.	6 to 52 grs. 18 months.	Tubercular Pleuro-Pneumonia (left base), acute. Spread to right lung.	Improved greatly; lost 5 and then gained 3 lbs. Iodoform "always stops cough."	Still ailing, but local condition almost quiescent.
4.	6 to 16 grs. 2 months.	I. Stage. Both lungs chronic.	Gave up iodoform because of gastric upset.	
5.	6 to 12 grs. 2 months.	III. Stage. Both lungs; severe pyrexia; fistula.	Expectoration gone. Appearance improved; lungs the same.	Larynx attacked, and dies 12 months later.
6.	6 to 12 grs. 2 months.	I. Stage. Both lungs. Has progressed since first seen.	Gained $4\frac{1}{2}$ lbs. Dyspnoea gone; lungs dry.	
7.	6 grs. 3 months.	III. Stage. Both lungs. Began as Left Pleurisy.	Cavities at both apices have dried and contracted; much general improvement.	Got quite strong. Returned to work for 18 months; now, 3 years later, recovering from another attack.
8.	6 to 12 grs. 5 months.	Larynx and right apex.	No improvement.	
9.	6 to 20 grs. 18 months.	I. Stage. Both lungs. Hæmoptysis $2\frac{1}{2}$ years ago.	Improved considerably.	For a year considered himself well, though husky; then came to me again with advanced laryngeal and nasal tubercle, and dies septicæmic.
10.	6 to 12 grs. 3 months.	I. Stage. Both lungs.	Much improved, locally and constitutionally.	Six months later improvement well maintained.

Of these ten patients five benefited very greatly, two improved slightly, and three were much the same during the period they were under treatment. It must be remembered that, except in one instance, these patients had no climatic or hygienic advantage, and nine of them continued to earn their livelihood, which, if not of a sedentary nature, has been one obliging them to spend the day indoors in not the best of atmospheres. They differed from hospital patients only in being better housed and better fed.

Of my in-patients at the Queen's Hospital during the past two years, I can only select six as fair samples of treatment with iodoform. All of these took the drug regularly for six weeks except one who, admitted in the last stage, died at the end of three weeks. Three of the other five suffered from nervous symptoms due to action of the drug, necessitating its omission. In one of these, in whom the symptoms arose when he was taking a daily dose of 32 grains, an acute toxic retrobulbar neuritis occurred, producing double complete central scotomata which did not thoroughly clear up for three months.\* His other symptoms of poisoning were typical of other cases—viz., a peristent taste of iodoform in nose and mouth, furred tongue, diarrhœa, giddiness, faintness, depression and irritable drowsiness, constant grumbling, restlessness, hallucinations, tingling and numbness of legs with increased knee jerks. I have also noticed a temporary aphasia, great

\* Reported by Mr. Priestley Smith in the *Ophthalmic Review*, April 1893.

excitability necessitating a male watcher, and a curious condition allied to alcoholic delirium, to wit, imagining little pigs and other curious animals and things upon the bed. But all these symptoms are purely temporary and cease soon after the omission of the drug, leaving no trace behind them. Nor do they appear to militate against the improvement, local or constitutional, of the patient.

All the five were in advanced stages of the disease and they all improved ; two very greatly indeed, for in one a large, probably tubercular, tumour in the abdomen completely vanished, whilst the lungs underwent considerable improvement ; and in the other, cerebral symptoms, which I diagnosed as tubercular meningitis, entirely cleared up, the patient gaining fifteen pounds in weight in two months, and losing nearly all signs of pulmonary mischief though he was admitted with a hectic temperature ranging from  $95^{\circ}$  to  $104^{\circ}$ .

They all commenced with a daily dose of 6 grains, which was quickly increased to an average maximum of 26 (in fourteen days) at which amount the treatment was continued.

Lastly, I should like to say a word or two as to the best method of administration. I believe it to be always safe to begin with 2 grains t.d.s., provided you warn your patient of the possible ill effects which may ensue. They will very rarely occur and if they do will come on gradually. The persistent odour and slight anorexia or an immature "bilious attack" are the most likely events. If your patient has difficulty in steadily taking

this small dose it is useless to persevere any further with the drug. Some advise in this event recourse to hypodermic injection, but, apart from its difficulty, I fail to see why such a symptom of constitutional saturation as persistent odour should not arise as quickly with this method as when the drug is given by the mouth. This opens up another point. If alimentary administration can produce such incontrovertible evidence of constitutional saturation as the nervous symptoms (*e.g.*) indicate I fail also to perceive why any better results can be obtained by hypodermic medication than by pills, though the superiority of the syringe is strongly upheld by some.

Having satisfied yourself by three or four days' treatment that the daily dose of 6 grains can be well borne, order it to be increased by 2 grains every other day till 30 grains are reached. Keep the patient to this daily dose for at least three months and at a somewhat lower level (should all signs of activity have vanished) for three months longer. But be very chary of dropping the drug altogether. I never *initiate* its disuse; and if the patient does not strongly object I always advise its continuance for a year. In fact, as I have before stated, I fall back upon the much greater experience we have of syphilis and its remedies and endeavour to guide my steps in accordance with this.

I have usually limited myself to a maximum dose of 30 grains a day, because Sternberg (*Amer. Jour. of Med. Sci.*, April 1883) showed that 30 grains of iodine should be sufficient to prevent the development



of various test organisms he employed in a man of 160 lbs. ; because iodoform contains 97 per cent. of iodine ; because it is decomposed in the body ; and, lastly, because its excretion is slow ; so that a daily dose of 30 grains should ensure the maintenance of 30 grains of iodine in the body. But Sternberg did not experiment with the bacillus of tubercle, which we know possesses a very stubborn vitality, so that the dose may be quite inadequate in its case. Moreover, there is no proof that iodoform's beneficial action is due to its direct destruction of the bacillus any more than there is a proof that mercury cures syphilis by its direct poisonous action on the syphilitic germ.

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